Southwestern Medicine

OFFICIAL ORGAN OF
ARIZONA STATE MEDICAL ASSOCIATION
NEW MEXICO MEDICAL SOCIETY
EL PASO COUNTY (TEXAS) MEDICAL SOCIETY
THE MEDICAL AND SURGICAL ASSOCIATION
OF THE SOUTHWEST

Volume XVI | February, 1932 | No. 2

ANNUAL SUBSCRIPTION $2 | SINGLE COPIES 25 CENTS

Entered at the Postoffice at Phoenix, Arizona, as second class matter.
"Acceptance for mailing at special rate of postage provided for in section 1103, Act of October 3, 1917, authorized March 1, 1921."

THE INFLUENCE OF HEREDITY, ENVIRONMENT, AND EARLY HABITS IN THE DEVELOPMENT OF PERSONALITY

MELDRUM KEPLINGER WYLDER,
M. D., F. A. C. P.
Albuquerque, N. M.

(Read before the Seventeenth Annual Meeting of the Medical and Surgical Association of the Southwest, held at Phoenix, Ariz., Dec. 3-5, 1931.)

It goes without saying that the most important job any of us have, or ever will have, is the raising of our children, but we often get so interested in our problem of making a living that we neglect our principal function in life and allow our children to grow up like weeds.

We want them to be healthy and strong, to be a little ahead of the height and weight tables for their ages, even though we are runts ourselves; but more than all that, we want them to be liked, to be pleasant, to make friends easily, to be systematic in their work and their play, to take good care of their toys, never to break or lose them; to stand up for their rights and not allow anyone to impose on them—and yet never to hurt anyone's feelings,—to make good in their studies and to excel in athletics. These are only a few of the common, almost universal, aspirations and ideals that parents have for their children. In short: we want them to develop this intangible something we speak of as "personality".

What is personality? I think that every one who has written on the subject has given at least one definition and when you see so many definitions, one thing is self-evident—that none of them are satisfactory. In the same way, you see hundreds of remedies recommended for asthma and it does not make much difference which one you use, the patient never gets over it anyway. While for itch only one remedy is recommended and it works. So, I am going to add to the problem by offering another definition. Personality is that something about you that makes people like, respect, and have confidence in you, or it makes people dislike, and have no respect for, or confidence in you. Thus, you see, personality is either negative or positive and we get all grades. No two personalities are ever alike. Should we develop a race in which strong, positive personalities were the rule, our army would be all generals and there would be no one to police the camp.

What factors enter into the formation of personality? And what are the deciding influences? Whether heredity or environment is of the greater importance, will never be settled. From the very beginning of our existence, hereditary tendencies are being constantly influenced by environment. Swift[1] says, "A certain original endowment is inherited but this is not personality. It is the basis, the foundation on which personality is built. The moment a child is born the environment begins to act on his rapidly developing plastic nervous system, conditioning certain responses and establishing certain types of behavior. These reactions to the stimuli of environment create the personality." The influences of both heredity and environment are at work constantly and you might just as well ask a gardener which is the most important, the seed, the soil, the water, or the climate. All are necessary; all must be right to produce the best products—neither good seed in poor soil, nor poor seed in good soil—and then, it must be watered and tended. The nights must not be too cold nor the days too hot and, if any one factor fails, the whole thing fails.

I think Kirkpatrick[2] sums it up pretty well in these words: "From the individual standpoint, heredity should neither be ignored as of no importance nor yielded to as inevitably fixing one's destiny. Instinctive and hereditary tendencies are the roots from which the physical, mental, and moral life develops. Some individuals develop more rapidly and

to a greater degree than others. All are of the same human characteristics but each makes himself out of his environment. Some cannot go as far as others in certain directions, nor as easily, but no one has ever exhausted his possibilities of development."

The practical problem is to expend our efforts on the useful characteristics that we do possess in the greatest degree. Whether we inherit such traits as honesty, dishonesty, selfishness, industry, laziness, or jealousy, from our parents, is impossible to state; however, we see such traits running in families. You often hear such remarks as this: "He can't be believed; his father was an awful liar."

Karl Pearson[3] says: "We inherit our parents' tempers, our parents' shyness, our parents' consciousness and ability, as we inherit their stature, form, and standing." While, on the other hand, Henry George[4] says: "The influence of heredity, which it is now the fashion to rate so highly, is nothing compared to the influences which mold the man after he comes into the world." These authors are quoted, not for the purpose of settling the question, but rather to emphasize the fact that the question has not been settled.

I think that we, as parents, teachers, and physicians, are apt to mislead ourselves by using heredity as a cloak and not analyzing the question and getting all the facts. An exasperated teacher who has failed to put over an illustration in such a way that the child gets it, instead of admitting her own failure, says: "Oh, he is dumb—his father and mother are both morons." A tired, exhausted mother, after one of her children has exhibited a temper tantrum (when arousing his interest in something else might have detoured him around it instead of through it) finds solace in saying, "You can't expect anything more from him: he's just like his dad; his whole family are like that." Gluck[5] emphasizes this danger of excusing our failures by blaming them on the ancestors, in these words: "Considerable mischief can be done to childhood through an unwarranted emphasis upon the hereditary burden that might be present if one ignores that caution which our limited knowledge calls for." When a parent or teacher sees in the behaviour of a child merely the reflection of something that was characteristic of some one or more of its ancestors, the immediate determining factors are apt to be ignored. The problem is apt to be viewed as a problem in predestination, which usually leads to the attitude of despair and failure instead of calm and endeavor to correct the situation. Moreover, the attitude of the anxious parent or exasperated teacher is bound to add its deleterious effects on the personality of the child to whatever factors may be already present.

The difficulty of evaluating the infuences of heredity and environment is well illustrated by Thom[6] in a pair of twin boys. The one was dull, listless, and apathetic. He took no notice of his surroundings, while the other was alert, active, his eyes sparkled, and he noticed every movement or noise; he cried a great deal if not taken up and had temper tantrums. These were identical twins, same heredity and in exactly the same room, but the environment was exactly opposite, for the family, friends, and neighbors always noticed, played with, and took up and exhibited the bright, alert, and nervous baby, thus exaggerating his already nervous tendencies, and they ignored entirely and took no notice whatever of the dull and apathetic chi'd, depressing him further and further into his lethargy. Thus you can realize that no two individuals can ever have exactly the same environment.

Aside from heredity and environment, we are what we are, on account of habits. When we were born, we were born without habits and we began at once to acquire them. Some one has said that we spend the first three years of our lives forming habits and the rest of our lives trying to break them. Whether the habits that we form are to be helpful or hurtful depends almost entirely on the training we receive. The earliest experiences of a child are the most important because paths are made by stimuli. These paths follow a course involving psychic, physical, and nerve mechanism associated with endocrine reaction. Such paths are most sensitive and the obliteration of a once well-established, psychic - physical, nerve - endocrine path is impossible. We might imagine every impression from the first to the last, though they number billions, to have created paths from without inward. Associated with these paths are all possible degrees of emotion. The repetition of any stimulus deepens and broadens these paths and they become fixed and thus, at a very early age, we acquire habits of punctuality, regularity, obedience, honesty, integrity, industry, and thrift; of content and satisfaction, if the habits we form are of the most desirable kind. On the other hand, they may be just the reverse.

The routine of the simplest existence constantly makes demands upon the habits that we acquire in early life; the time we get up, the daily bath, our forms of exercise and recreation, the extent to which we indulge our appetites, our attitude toward our associates and those with whom we come in

contact. We are scarcely aware that our decisions are largely guided by that unseen force called habit.

Habits associated with character are of the greatest importance. I may mention honesty. A child who acquires the habit of honesty saves himself many problems. He does not have to ponder over the question of whether to tell the truth or not. He will tell the truth and do the square, honest, manly thing unhesitatingly. We are all hero worshipers. We all heard, among the first stories that we can remember, the one of George Washington—the boy who never told a lie. I heard it, and so did you, and I well remember the first time I visited Mount Vernon. As I stood with bared head before that most sacred American shrine, that little brick enclosure that bears this simple inscription: "Within this enclosure lie the remains of General and Mrs. George Washington.

The first thought that came to my mind was not of the great general, military genius, statesman and Father of his Country, but that he was the boy who never told a lie. And it was, in all probability, on account of this early formation of the right kind of habits, the sterling habits that go to make up character and personality, that he became the great man that he was.

Habits make demands for certain kinds of activity. They rule our thoughts. Habits that go to build the desirable type of personality must be acquired early. They are not like long trousers, to be put on at a certain age. They are matters of growth and development. The mental characteristics and moral qualities that go to develop success or failure are, to a large extent, matters of habit. Ambition, thrift, persistency, fairness, cleanliness, laziness, selfishness, carelessness, distinctiveness, and numerous other traits of character that go to make up personality, are not inherited. Tendencies in their direction may be, but, unless they are developed by stimulation they remain impotent in their effect on personality.

The doing a thing over and over gives no positive assurance that the habit will be long continued, and it will not be unless the child gets, as a result of the accomplishment, some emotional satisfaction. The amount of emotional satisfaction the child obtains from learning proper habits of eating, eliminating, and obedience, is derived to a great extent directly from those whose approbation is most desired. All children have not the same desire for praise. They are not all well endowed with that personality trait which would make them want to please. Other incentives must be used. Reward, praise, blame, and punishment must all be used at times in an effort to develop for the child a personality that will help him to assume the responsibilities of life. The child should be led, not pushed. Too often it is the emotions, and not the intellect, that impede the formation of proper habits.

Encouragement is the most powerful instrument we have. Encouragement is something we all need. Usually the best physician is the one who succeeds best in encouraging his patients. By encouraging a child you can make him do wonders. We have all read some of the letters of the late Colonel Roosevelt to his boys; they were published after his death. We are struck in a number of letters with the father's way of bringing out the best in his children. His conjuring up, by a mighty expectation, qualities which undoubtedly were in the children anyway, and bringing them to the fore by a mighty taking for granted that amounts almost to hypnotism. "I know you are studying hard," he writes one son, though in the next letter he says he is puzzled by the low marks. He suggests an explanation himself, however, in no way reflecting on the boy's industry and ability. "I have faith in your energy," concludes a letter that might have been concluded in another way, "in your perseverance, your ability and in your power to force yourself to the front, when you have once found out and taken your line." And again, "You have gone in with a serious purpose of doing decently and honorably, of standing well in your studies and getting the respect and liking of your classmates so far as they can be legitimately attained." All through these letters of Theodore Roosevelt to his children, breathes the spirit, "I believe in you". The children of such a father could not fail. We cannot but contrast that with the other parental attitude, altogether too common, that the child is always to blame, mostly in the wrong, being a child. The parental power of suggestion goes a long way toward pulling down or building up the personality of the child yet in the making.

Undue concern on the part of the parents often causes marked changes in the personality of a child. After a serious accident or illness which has placed him in danger and caused anxiety in the household, it is difficult for parents to control him wisely. They may cease to exert authority, fearing harm will result, and it takes only a short time for a child to learn that with his symptoms he can control his environment. Over-solicitude often defeats its own ends by creating such an intense emotional situation that the original difficulty is only exaggerated. The attitude that many parents take of expecting

undesirable conduct and of inability to cope with it, has a strong influence in children. If it is constantly suggested that Bill can not eat what other children eat, or that he will not stay in the yard, there is no cause for surprise if Bill runs away at every opportunity. Children want attention and will attain it somehow.

It is not my intention to leave the impression that the influence of the home is destructive to the personality of the child, but rather to emphasize the tremendous influences, constructive or destructive, wielded in the home and the terrible responsibilities and opportunities that are on the shoulders of parenthood. Since, as we have shown, most habits are formed and fixed in early life, before school age, I do not believe that these children with undesirable habits and delinquent tendencies are the product of an irreparable past over which there is no control. They are largely the result of the environment in which they grow up, and the dominating feature of that environment is always the parent.

The formation of personality, then, depends more than anything else, and more than everything else, upon the parents, and I think that Abraham Lincoln summed it up better than it has ever been summed up by anyone else when he said: "All that I am and all that I ever hope to be, I owe to my sainted mother."

BIBLIOGRAPHY

1. Swift, Edgar James: Psychology of Childhood, D. Appleton & Co., 1930, p. 45.
2. Kirkpatrick, E. A.: Fundamentals of Child Study, Macmillan Co., 1922, p. 29.
3. Pearson, Karl,: quoted in The Trend of the Race, by S. J. Holmes, Harcourt Brace, p. 98.
4. George, Henry,:quoted in same, p. 99.
5. Gleuck, Bernard,: Mental Hygiene, Vol. 8, No. 3.
6. Thom, Douglas A.: Everyday Problems of the Everyday Child, D. Appleton & Co., 1930. p. 8.

JAUNDICE

FRED H. KRUSE, M. D.
San Francisco, Calif.

(Read before the Seventeenth Annual Meeting of The Medical and Surgical Association of the Southwest, held at Phoenix, Ariz., Dec. 3-5, 1931.)

To the invaluable knowledge to be gleaned from clinical and roentgen examination of an individual with jaundice, Mann and his associates have emphasized the importance of two great discoveries in their study of the liver, that have aided the clinicians in their classification and interpretation of the various types of jaundice and in applying to practical purposes, both for diagnosis and treatment of these conditions, the newer knowledge of liver function.

These two fundamental assumptions are:

1. That bile pigment (bilirubin) is formed outside the liver and is brought to it for excretion. Until recently it was generally held that bile pigments are formed in the liver from hemoglobin liberated on the disintegration of red blood corpuscles. The theory of an extrahepatic origin of bile pigment has become firmly established in present-day physiology.

2. The importance of the carbohydrate function of the liver; namely, that the liver as a vital function stores glycogen (as discovered in 1857 by Claude Bernard) and immediately after the removal of the liver (or theoretically by its destruction or paralysis) the blood sugar decreases progressively. When it reaches a certain rather definite limit, of from 0.06 to 0.04 per cent, a characteristic group of symptoms appears, and, if the decrease is allowed to continue, death soon ensues.

On account of the above conclusions and the light shed by similar work in relation to the liver and jaundice, the tendency has been to simplify our classification of jaundice, clinically, into three groups: (1) obstructive jaundice, including cases in which there is occlusion of or obstruction in the extrahepatic biliary passages; (2) intrahepatic jaundice, including cases where the diseased process is within the liver, affecting the hepatic parenchyma as a whole, or limited to the bile passages or the cells of the liver itself; and (3) hemolytic jaundice, associated with increased destruction of blood cells and subsequent increase in the level of bile pigments in the blood (excessive bilirubin production).

We might pause here to summarize briefly, under these three groups, the various more common diseases and abnormal conditions arising in the body which cause jaundice.

It has been variously estimated that the percentage rating of the above three groups is about as follows: obstructive jaundice cases, 70 to 80 per cent; intrahepatic jaundice cases 20 to 25 per cent; hemolytic jaundice cases, 5 to 10 per cent, of all.

1. Obstructive Jaundice: In a survey of such cases of jaundice at the Mayo Clinic recently, carcinoma accounted for about 25 per cent of these; malignancy of the pancreas was the most common single cause, and tumors of the gallbladder and bile ducts taken together were about half as common. Metastatic lesions and general carcinomatosis accounted for about 7 per cent of all cases of jaundice. Of the benign lesions, stones in the common bile duct were responsible for 20 per cent of all jaundice cases; and stricture of the common or hepatic ducts, 10 per cent. This stricture occurs most frequently as an aftermath of operation, but

may develop spontaneously from inflammatory causes, or may be of neoplastic origin.

Compression of the ducts may occur from tumors in any of the neighboring organs, or in the liver itself, or from aneurysm; or kinking or torsion of the ducts may occur from ptosis or abdominal tumors, such as the pregnant uterus, and so forth. Often pressure and obstruction of the ducts in the liver occur from abscesses (such as amebic abscess), tubercles, gummas, hydatid cysts, and primary and secondary new growths in the liver, which may give true obstructive jaundice, and, in addition, give rise to a hepatitis or disease of the liver cells, thus adding the intrahepatic element to the jaundice, to confuse the picture.

Chronic or hemorrhagic pancreatitis may be the cause of the jaundice in association with a diseased gallbladder.

In a recent review, twenty cases of jaundice were due to stricture of the common duct, and nineteen of these had had previous operations on the biliary tract. The degree of jaundice as shown by the serum bilirubin or icterus index was not great.

2. Intrahepatic Jaundice (Toxic and Infective): This type of jaundice occurs from the results of bacterial infection or the damage done by some chemical, drug, or other toxin, to the liver tissues and cells. The pathology varies from simple cloudy swelling and congestion to acute inflammatory and suppurative processes or actual dissolution and destruction of extensive areas within the liver parenchyma, termed hepatolysis. The disease groups range in intensity from a simple catarrhal jaundice to the destructive processes of acute yel'ow atrophy. One of the most common forms is a hepatitis (of various degrees). All cases of gallbladder disease have an associated hepatitis, mild or severe in intensity. As the damage increases or persists, there develops chronic cholangeitis, suppurative cholangeitis, and suppurative hepatitis, or liver abscess.

As I have said above, we most commonly think of intrahepatic jaundice as being infective or toxic in origin.

The infections or bacteria reach the liver by the blood stream, by the lymphatics (especially from diseased gallbladders), and by the ascending route from the duodenum and ampulla of Vater. These bacteria produce a hepatitis. The hepatitis may occur first and lead to a secondary gallbladder infection, but, in any event, the gallbladder once infected leads to repeated infective shocks of the liver, producing a hepatitis, after a period of cloudy swelling and congestion, and is the most frequent cause of chronic cholangeitis and, finally, suppurative cholangeitis, all producing jaundice. Bacteria from the appendix or other foci of infection in the abdomen, from foci in the head (nose and throat or teeth), from a septicemia, may be etiological factors, as well as in association with pneumonia or typhoid fever; but infection of the liver is rendered much easier of accomplishment by local disease of the biliary tract.

Indeed, aside from virulent infection, it is doubtful if infection of the biliary tract ever occurs in the absence of local biliary disease, since the free flow of bile is usually considered an effective preventive of biliary infection. In the great majority of cases, suppurative cholangeitis is definitely related to local disease of the biliary tract, such as gallbladder disease or acute impaction of stones in the ducts; and this kind of cholangeitis of varying grades not infrequently follows as a sequel of cholecystectomy. In either a latent or severe form, it may antedate surgery. It more commonly occurs, however, as a result of a residual hepatitis.

In hepatitis, fever may be constant or intermittent; chills may be irregular. Jaundice is practically always present, variable in degree. It may vanish with periods of drainage and recur, but is generally slight or moderate only. Often there is a dull aching in the region of the liver, which is enlarged and continues to enlarge. A leukocytosis develops with the febrile periods, and generally is absent in afebrile periods.

Toxic jaundice has developed from certain internal poisons (toxins of endogenous origin) as in pregnancy and toxic hyperthyroidism (in both cases frequently resulting in acute yellow atrophy of the liver); but chiefly from intoxicants or tissue poisons, such as phosphorus, arsenic (especially Fowler's solution and salvarsan), atophan or cinchophen and its various compounds, chloroform, picric acid (and certain other nitrophenols), yatren, trichlorethane, trinitrotoluene, snake venom, lead, and phenylhydrazine. The liver has suffered from the above in various degrees, from an apparently mild toxic hepatitis or catarrhal jaundice, with recovery, to a serious destructive process with necrosis and dissolution of liver cells, better perhaps described as a hepatolysis. In the more destructive or extensive cases of hepatolysis, yellow atrophy results.

In this intrahepatic classification we find cases of biliary cirrhosis, even to the hypertrophic or Hanot's cirrhosis, as well as the jaundice of syphilis. Abscess of the liver from various septic foci, and the tropical or amebic abscess may lead to a secondary hepatitis and thus an intrahepatic jaundice.

3. Hemolytic Jaundice: The outstanding

disease entities to be grouped here are: (a) chronic hemolytic icterus, with splenomegaly (especially the familial form); (b) von Jaksch's anemia; (c) the icterus of pernicious anemia and related anemias and that associated with certain parasitic diseases; and (d) icterus neonatorum.

While a large percentage of cases due to the above disorders present, clinically, features that render them easily distinguishable, frequently they do not; and it is almost impossible by history, physical and roentgen-ray examination to determine the origin of the jaundice, particularly if we are unable to secure an intelligent and exact history.

Again I should like to emphasize the importance of an exact history in reference at least to the presence or absence of pain and its character when present. However, while it must be recognized that the distinction of "painless jaundice" and "painful jaundice", both as to history of onset and course, may be the deciding factor for diagnosis—there are enough exceptions, both ways to the general rule, to lead us to look for other evidences confirmatory of the impression gained from the history and physical examination.

Judd and Marshall, in reporting a series of 1608 patients with stone in the common bile duct, found in forty (2.4 per cent) no pain occurred at any time in the course of the disease; while, on the other hand, I have seen cases of cholangeitis or hepatitis occurring several months after removal of the gallbladder, with pain simulating that of the colic of gallstones, and have observed cases of jaundice with recurrent pain in carcinoma of the gallbladder.

Since these various cases of jaundice have very different therapeutic indications, and yet when first seen cannot be differentiated by the ordinary examination, we must have clearly in mind other methods of procedure that may give us information and points essential to a working classification. These other procedures or tests, grouped according to the type of information yielded, are:

(1) Tests for the quantity of bile reaching the intestines, as determined by siphonage of the duodenal contents (duodenal drainage).

(2) By the same method, determination of the presence or absence of the pancreatic ferments.

(3) Studies of liver function in respect to excretion, as determined by the van den Bergh reactions, quantitative bilirubin, icterus index, rose bengal dye test, the Graham hepatic-function test as determined by the elimination of phenoltetriodo-phthaleinsodium, and, finally, quantitative urobilinogen estimations.

(4) Studies of liver function in respect to the handling of carbohydrates or the metabolic-function test as determined by modified glucose-tolerance test, and the galactose-tolerance.

As to the first two procedures, the duodenal and pancreatic studies, their clinical usefulness and significance is apparent in assisting in determining both the degree and location of the block, and I will not herein enter into fuller discussion of their limitations and possibilities.

As regards the tests for liver function, there is considerable disagreement among those actively engaged in their development in the laboratories, and, among clinicians at large, I have found the utmost confusion as to their significance and hesitation in placing any dependence upon their clinical usefulness.

And yet, in the light of some recent reports and observations, and by comparative studies where these tests have been made in groups of cases in which the pathology has been determined at operation or autopsy, undoubtedly their clinical usefulness has been demonstrated.

BILIRUBIN

In order to understand and interpret the excretion tests of liver function, such as the icterus index, van den Bergh, urobilinogen, and the dyes (rose bengal), we must have some conception of the physiology of bilirubin formation and excretion, since, in cases of jaundice, these tests are expressions of the bile pigment (bilirubin) concentration in the blood stream or else, as in the instances of the dyes, their elimination by the liver parallels that of the bile pigment. Mann has pretty conclusively demonstrated the extrahepatic origin of bilirubin in mammals.

We know, from experimental observation and the development of the van den Bergh tests, that the bilirubin carried to the liver from its various sites of origin in the body, is different from that leaving the liver in the bile and therefore that the liver cells are the initiators of this change in physical or chemical state. Utilization of this knowledge as a diagnostic aid has been of most material assistance in the differential classification of types of jaundice.

We might think of the freshly formed bilirubin in the tissues of origin as nascent bilirubin. It is produced extrahepatically at a very rapid rate and travels to the liver by the blood stream. Perhaps, as suggested by Elton, it exists as a suspensoid colloid, and, as such, cannot permeate the cells of the liver parenchyma. Hence, certain cells of

this parenchyma (possibly the Kupffer cells in mammals) act as acceptors and change its state so that the real parenchymal or polygonal cells may complete a final chemical change. Perhaps, as suggested by Elton and others, the bilirubin leaving the liver is a crystalloid. Thus the body would have three forms of bilirubin constantly to deal with: (a) a suspensoid colloid, carried to the liver in the blood; (b) an unstable, changing product in the liver cells; (c) a crystalloid or soluble form leaving the liver by the bile channels.

VAN DEN BERGH REACTIONS

At anv rate, we generally agree that nascent bilirubin, as it travels to the liver, is changed and is different in character from the bilirubin of the bile excreted from the liver. In diseased conditions, the orderly process of change and elimination is interfered with and the blood stream becames flooded with all kinds of bilirubin, as indicated by the van den Bergh reactions, which may be most helpful if interpreted correctly and the limitations of the tests are recognized, but are usually most confusing and misleading unless properly understood and correlated with other evidence.

If the common duct is ligated, obstructive jaundice ensues, due to flooding of the blood stream with the excretion form of bilirubin,-crystalloid (?) state.

The van den Bergh reaction is then direct positive. If the liver is removed, jaundice ensues, due to accumulation in the blood of nascent bilirubin—suspensoid colloidal (?) form—or in man, with the liver intact, in hemolytic diseases due to extensive nascent bilirubin production and accumulation, too great for the liver to handle. The van den Bergh reaction is then indirect, demonstrated usually by bringing the bilirubin into alcoholic solution.

These are the only clear and uncomplicated forms of the reaction, and occur only when the liver parenchyma is functioning normally. When the jaundice is prolonged and the liver cells are overwhelmed by intense pigmentary congestion, or if they are affected by disease within the liver, the blood is then flooded with all kinds of bilirubin and the mixed (biphasic) and confusing reactions result, none of which taken alone are diagnostic of any disease entity.

A schematic summary of these conditions and their most likely variations is helpful as a graphic aid in their interpretation. (Figures 1 and 2).

THE ICTERUS INDEX

The icterus index may be defined as a physical measurement of the yellow color intensity of serum. The determination is based, not upon the amount of bilirubin in the serum, but upon the assumption that the color of the serum is largely due to bilirubin.

The ingestion of eggs, oranges, and chlorophyl-containing vegetables, in children, and of carrots, in both children and adults, may increase the index proportionately. The normal icterus index lies between 4 and 6, with an average of 5. As clinical icterus has never been present with readings below 15, and is rarely absent with readings over 15, a reading between 6 and 16 indicates a zone of latent icterus, seen so frequently in gallbladder disease without jaundice. In hemolytic diseases, the icterus index will vary from 6 to 16, or within the zone of latent jaundice. In these diseases, however, the quantitative serum bilirubin will run quite high, entirely out of proportion to the low icterus index, indicating the presence of nascent bilirubin (the so-called colloidal suspensoid), which does not color the serum highly, since it is theoretically not in solution. Beyond the reading of 16, in true icterus, the final, or crystalloid, form of bilirubin formed in the liver and in the blood imparts more intense color, and, as the icterus mounts in intrahepatic or obstructive jaundice, the quantitative bilirubin and icterus index increase proportionately, and a high icterus index indicates a high quantitative bilirubin level.

Relatively high bilirubin values (quantitative) in and near the normal zone of the icterus index were found in sera from patients with familial jaundice and pernicious

VAN DEN BERGH REACTION

Direct Positive Direct Immediate	Indirect Direct Negative Direct Delayed
DIAZO REACTION PROMPTLY	DIAZO REACTION LATER by adding oxygen or by manipulation 1. Standing 2. Heating 3. Adding Alcohol
Typically in early Biliary Duct Block	
OBSTRUCTIVE JAUNDICE	HEMOLYTIC JAUNDICE

Either of these when prolonged produce

BIPHASIC REACTION

Mixture of all kinds of bilirubin

Any intrahepatic damage or congestion
Catarrhal Jaundice
Hepatitis
Yellow Atrophy
Toxic Cirrhosis

Fig. 1. Schematic illustration of the van den Bergh reaction in different types of jaundice.

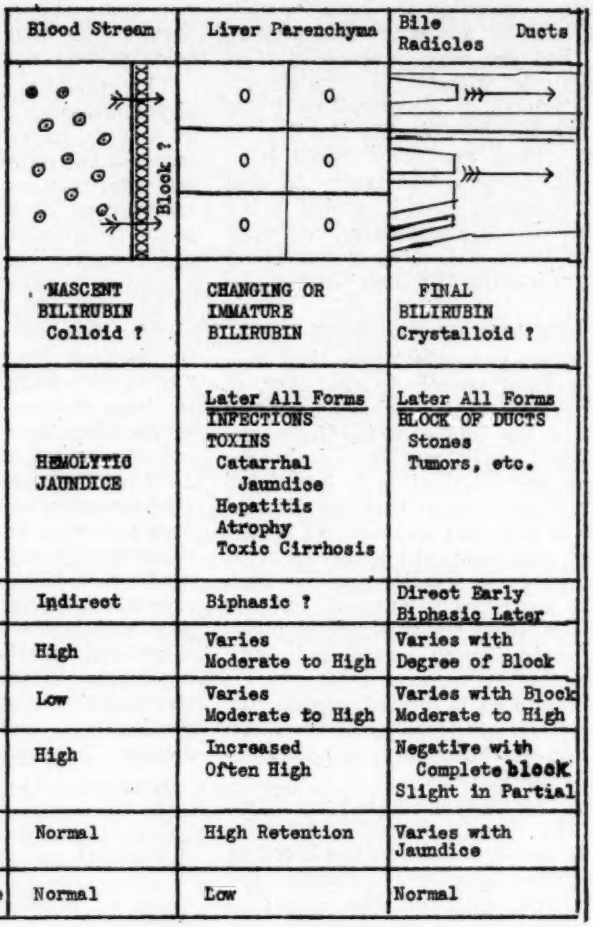


Fig. 2. Schematic illustration of the formation of bilirubin and its relation to various tests.

anemia, in placental blood, and in patients suffering from trauma and wounds.

Very high readings in the presence of evident and well-marked clinical jaundice suggest the obstructive type, this being especially true in carcinoma of the pancreas obstructing the ampulla; but parallel readings may be found in catarrhal jaundice and acute yellow atrophy. In jaundiced cases with a definitely increased icterus index, the dye function tests (such as the rose bengal) are generally superfluous, since we know the dyes will be retained if the bile cannot escape. Withal, the icterus index is a most useful functional test of the liver and is of value in demonstrating latent jaundice; in establishing a quantitative index for the degree of jaundice in the various toxic or infectious types; in estimating the degree of retention of bile, in obstructive jaundice, and, finally, in detecting the clearing or subsidence of a deep jaundice; thus distinguishing a benign catarrhal form from a jaundice due to a malignancy, which remains stationary or increases.

UROBILINOGEN

The bile pigments that pass through the liver and, via the bile passages, enter the intestine, are reduced in the large intestine by putrefactive organisms into a pigment which is partly excreted with the feces (stercobilin) and partly reabsorbed into the portal circulation (urobilin). It is believed that most of the urobilin passes back to the liver, where it is again utilized. In insufficiency of the liver, urobilin enters the systemic circulation in varying amounts and may be readily demonstrated in the urine. This, of course, depends upon whether or not bile pigments are entering the intestine. If there is a complete block of the bile channels (generally at the ampulla) no bile enters the intestine and there is no production of urobilin to enter the urine. Most of the cases of ob-

structive jaundice do not have a complete block, and, hence, increased amounts of urobilin in these point also to liver damage or insufficiency. Relatively high urobilin readings are also to be expected in cases of intrahepatic jaundice, due to the liver insufficiency brought about by the character of these diseases.

In hemolytic jaundice, the presence of urobilinogen in the urine is due to the excessive amounts of bile pigments that the liver puts out, and the consequent over-flooding of the liver with both nascent bilirubin, brought by the blood stream, and urobilin, so that the liver is unable to handle all that is brought to it, resulting in the excess being eliminated in the urine.

Urobilinogen is a chromogen of urobilin formed by reduction of urobilin. The two are otherwise identical, and the demonstration of either in the urine has the same significance clinically.

The test for urobilinogen is more delicate than the fluorescent test for urobilin, is quite simple, and lends itself readily to quantitative estimation.

It is to be remembered that the intestine is the sole source of urobilinogen.

All conditions in which urobilinogen is

DISEASES	ICTERUS INDEX	VAN DEN BERGH	BILIRUBIN	ROSE BENGAL	GLUCOSE TOLERANCE	UROBILIN-OGEN
Normal Serum	3 to 6	No Reaction or Indirect	0.1-0.35mgm.	Normal 20. 30.	Normal	0
Latent Jaundice	6 to 16	Varies with Type of Jaundice	0.4-2. mgm.	Generally Normal	Normal	0 to +
Hemolytic Jaundice	P.A.9-16(30) Fam. 15-60	Indirect (Direct Delayed)	1 - 16mgm.+	Generally Normal	Normal	++ +++
Hepatitis and Cholangeitis with Jaundice	Low ++ to High	Biphasic	1 mgm. to High	Mod.to High Retention with Degree Jaundice	Generally Low	++
Common Duct Stone Common Duct Stricture	Moderate++ to High	Direct+ (Early) Biphasic(Late)	4-30 mgm.+	Varies with Degree of Jaundice	Normal to Slight	0 in Complete Block
Liver Abscess	Low ++ to Moderate	Biphasic (Indirect)?	Normal to Low	Normal to Moderate	Generally Normal	0 ++
Salvarsan Jaundice	Moderate++ to High	Biphasic	4-30 mgm.+	Moderate to 100% Retention	Low	0 ++
Carcinoma of Liver	Generally Low ++ to High	Mixed according to Location	Low to 30mgm.+	Normal to 100% Retention	Normal Occasionally Low	0 ++
Carcinoma (Pancreas (Ampulla	Moderate to High+++	Direct + (Early) (Biphasic) Late	1-30 mgm.+	Slight to 100 % Retention	Generally Normal Often Low	0
Catarrhal Jaundice	Moderate to High+++	Biphasic (Direct if Early)	4-30 mgm.+	Moderate to 100%	Low	Early 0 +++
Acute Yellow Atrophy	High	Biphasic	High	80-100%	Low	0 ++
Chronic Yellow Atrophy	Moderate to High +++	Biphasic	Moderate to High	Moderate to 100%	Generally Normal to Slightly Low	0 ++
Portal Cirrhosis	Normal to Low	Biphasic (Indirect)	0.1-3.mgm.+	Moderate to 100%	Generally Low	0 ++
Hypertrophic Cirrhosis	Low to Moderate	Biphasic (Indirect)	1.-3 mgm.+	Moderate to 100%	Generally Low	0 ++
Toxic Cirrhosis	Moderate++ to High	Biphasic (Indirect)	1.-30mgm.+	Generally High Retention	Normal	0 +

Fig. 3. Tabulation of the various tests in different diseases.

eliminated in excess can be classified in two groups:

1. Diseases of liver and biliary tract (especially in parenchymatous change).

2. Hemolytic diseases.

Referring to the first of these, urobilinogen is found in largest amounts in acute and subacute liver changes, and in acute exacerbations of chronic processes. Thus, in catarrhal jaundice there are increased amounts in every case at the beginning and end of the disease; whereas, at the height of the disease, it may be low or absent, due to the more or less complete block at the ampulla. Also, in hepatitis, cholangeitis, liver abscess, malaria, and yellow atrophy, it may be relatively high. In slowly progressive chronic lesions, a high output is not constant. This is probably due to the ability of the liver to carry on its normal metabolic activity by compensatory growth of new liver tissues. This ability, and its effect on sugar tolerance tests, will be referred to later. Thus, in certain cases of cirrhosis, syphilis of the liver, Banti's disease, splenomegalies, and subacute conditions, such as cholecystitis and cholelithiasis, the readings tend to be low.

In summary, the value of this test is emphasized in painless jaundice, as urobilin is always present at some time in catarrhal jaundice; and in mechanical icterus, such as carcinoma of the head of the pancreas or ampulla, its total absence forms a marked diagnostic feature.

THE DYE TESTS FOR LIVER FUNCTION

In these tests, the dye selected is injected into the blood stream, and its concentration remaining after certain intervals of time, is observed. These dyes are eliminated from the organism by means of the activity of the liver parenchyma cells and are selected also because they are non-toxic and stain the serum sufficiently. They are eliminated in the bile. The two that have survived and are most widely used are:

1. Rose bengal (diiodotetrachlorfluorescein of the triphenyl-methane series).

2. Phenoltetraiodophthalein (sodium).
Iso-iodeikon (Graham's dye)

The latter has the advantage of allowing visualization of the gallbladder at the same time (cholecystography). After ligation of the common duct, in animal experimentation, there is no retention above normal for two or three days. The retention of the dye usually coincides with the first evidence of, or appearance of, bile in the blood—that is, two or three days following obstruction of the common duct, and generally reaches a maximum between the second and fourth weeks. It is obvious, therefore, that the chief cause of failure to eliminate the dye is damage to the liver parenchyma. While, in pure obstruction of the bile passages, the dye is retained as the bile backs up, it is not until the liver cells suffer from stasis congestion and infection following obstruction that the dye is retained in the blood.

Thus, in non-obstructive jaundice from toxins or infections, such as catarrhal jaundice and acute yellow atrophy, the dye retention is quite high, but will lessen as recovery takes place, returning to normal in such cases as catarrhal jaundice and often in arsphenamine icterus. On the other hand, in obstructive jaundice the degree of retention of the dye, if it is not given too late in the disease, is a material aid in differentiation between carcinoma (especially of the pancreas) and stone in the common duct. Associated with stone in the common duct, there is always a hepatitis and much more liver damage, especially early, than in carcinoma, and therefore a higher concentration of the dye in the blood serum.

The Graham dye, in patients with jaundice produced by stone in the common duct, rises rapidly to 30 to 60 per cent retention in 30 minutes, after the first few days of obstruction; whereas, in carcinoma (not terminal) the figures are more apt to be 10 to 25 per cent in 30 minutes.

Evarts Graham found, in cases either with or without jaundice, using the phenoltetraiodophthalein dye, that a high retention (50 per cent or more) indicates a poor operative risk. He likes to treat these cases with high carbohydrates, improved drainage and conservative measures, restoring the liver reserve and glycogen stores until the retention is not over 35 per cent.

In the University of California wards and clinics, we have used the rose bengal test chiefly, but have felt that in well-marked jaundice, the test is generally superfluous and will run parallel to the icterus index, indicating that the amount of liver damage (at least temporarily) varies according to the depth of jaundice. However, there are undoubtedly a few exceptions to this, but either dye is valuable in estimating the operative risk. Retention of rose bengal in the blood above 60 per cent at the end of 8 minutes and over 30 per cent after 16 minutes, is indicative of decreased permeability of the hepatic cells or of obstruction to the free flow of bile.

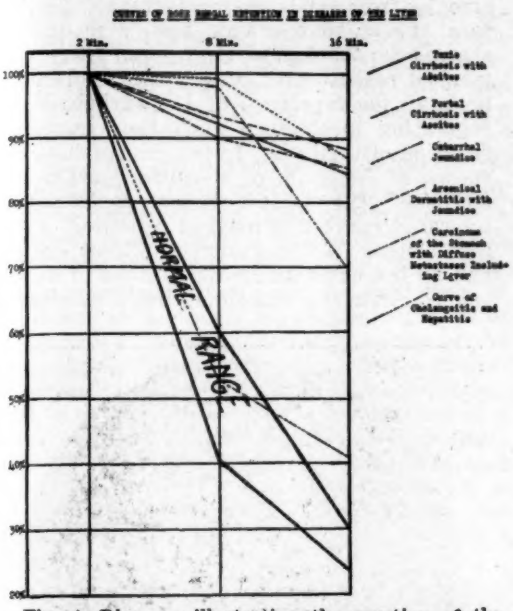
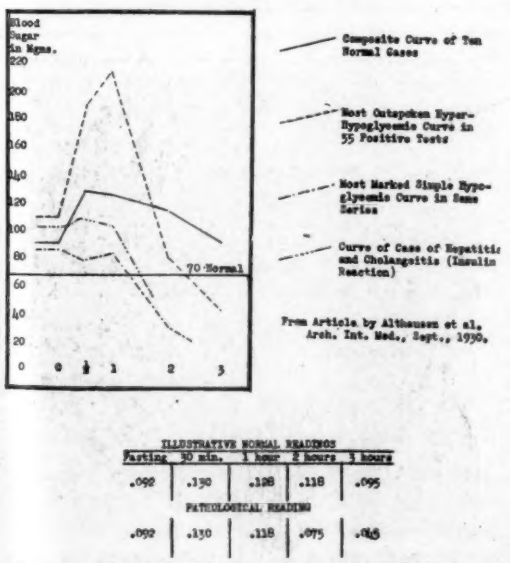


Fig. 4. Diagram illustrating the reaction of the liver to the rose bengal elimination test in various conditions, as compared to the normal range.

In non-jaundice cases, where the liver is suspected, especially those with ascites, since rose bengal is retained to a considerable degree in liver cirrhosis, and so forth, and is not in congestive heart failure or tuberculous peritonitis, a valuable aid in differential diagnosis is secured.



ILLUSTRATIVE NORMAL READINGS

Fasting	30 min.	1 hour	2 hours	3 hours
.092	.130	.128	.118	.095

PATHOLOGICAL READING

.092	.130	.118	.075	.065

Fig. 5. Chart of the modified glucose tolerance test, as developed by Althausen, showing the reaction in several conditions.

SUGAR TOLERANCE TESTS (Metabolic Test)

There are two notable sugar-tolerance tests:

(1) the modified glucose-tolerance test;
(2) The galactose-tolerance test.

The modified glucose-tolerance test, as developed by Althausen and others, is a simple modification of the dextrose-tolerance test, in which insulin and a large amount of water are given to the patient in addition to 55 Gms. glucose by mouth, and depends on the carbohydrate-metabolism-regulating function of the liver. Blood sugar readings are made in 30 minutes, 1 hour, 2 hours, and 3 hours after the glucose. A blood sugar level below 70 mg. per 100 cc. of blood at the conclusion of the test indicates a deficiency in the carbohydate-regulating function of the liver. It is not an excretion test and therefore does not always check with the rose bengal. It brings out latent functional impairment of the liver. In combination with a dye excretion test, it helps in indicating the amount of regeneration taking place in the liver and the functional capacity of this new tissue, and thus affords a valuable diagnostic aid in toxic cirrhosis of the liver.

THE GALACTOSE TOLERANCE TEST

In this test, pure galactose, 40 Gms. dissolved in 500 cc. of water, is administered orally, and the total quantity of galactose excreted in the urine in five hours is estimated. Outputs of 3.0 Gms. and more have been accepted as indicative of impaired hepatic tolerance. This lowered tolerance has been found positive only in the group of toxic and infective jaundice cases (intrahepatic jaundice) and has not been either so delicate or reliable in individual cases as the modified glucose tolerance tests.

T. L. Althausen, (Archives Int. Med. Oct. 1931) has called attention to the functional aspects of regenerated liver tissue as exemplified in a series of cases of toxic cirrhosis.

Figure 9 illustrates a section of liver showing marked fibrosis of irregular distribution, with an example of liver regeneration, the typical adenomatoid areas of hepatic cells. Figure 10 shows a single adenomatoid nodule of hyperplastic cells, with a great loss of the hepatic substance surrounding it.

In this case the liver had been badly damaged, but the new tissue was able to carry on the carbohydrate-regulating mechanism efficiently, as shown by a normal modified glucose-tolerance curve. However, there was marked retention of rose bengal, which was interpreted to indicate that in spite of regeneration the new hyperplastic nodules lack connection with bile channels and, hence, could not excrete the dye.

The illustrations of figure 7 show a marked destructive process in the liver (acute liver atrophy from cinchophen poisoning). In this case the sugar-tolerance test with insulin showed a pathological curve, and there was marked retention of rose bengal, indicating both an incompetence of the carbohydrate-regulating mechanism and the excretory function of the liver. In the case of the liver shown in figure 8, of so-called "obstructive cirrhosis", there was a normal insulin sugar-tolerance curve and marked retention of the rose bengal. At operation, the liver grossly had all the external characteristics of an advanced cirrhosis, was small, but a carcinoma

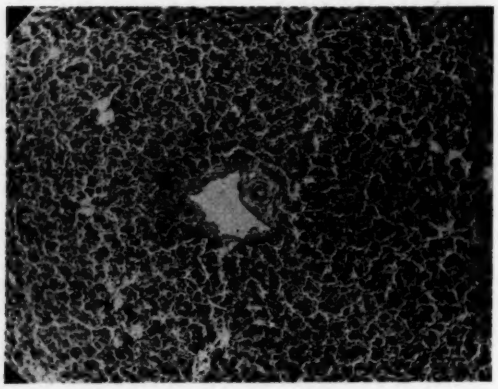


Fig. 6. Section of normal liver, showing histologic structure.

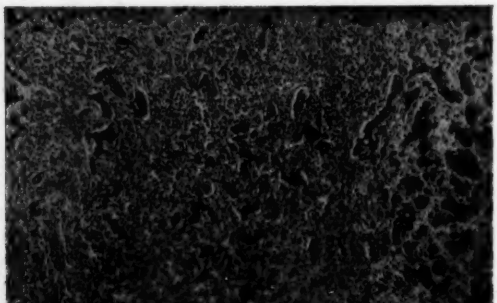


Fig. 7. Section of liver showing the changes produced by acute yellow atrophy (case of atophan poisoning).

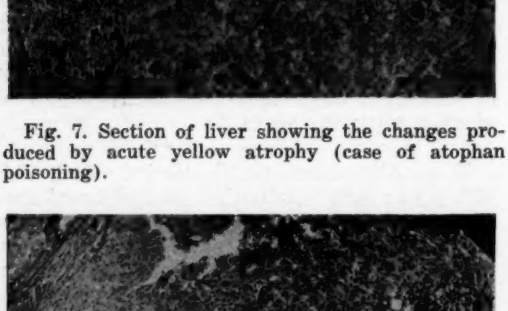


Fig. 8. "Obstructive Cirrhosis," due to bile block. Bile distended channels appear as dark areas.

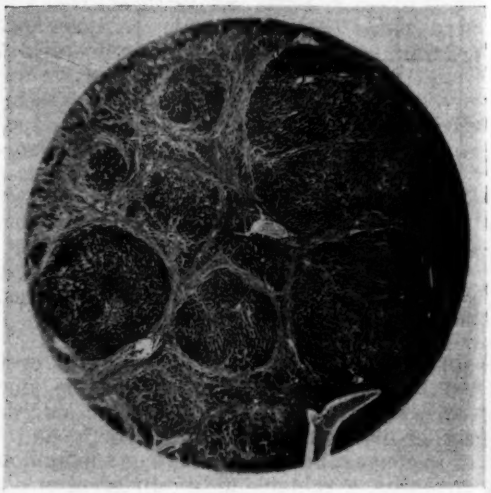


Fig. 9. Section of liver showing marked fibrosis, irregularly distributed, with regeneration in form of adenomatoid areas of liver cells.

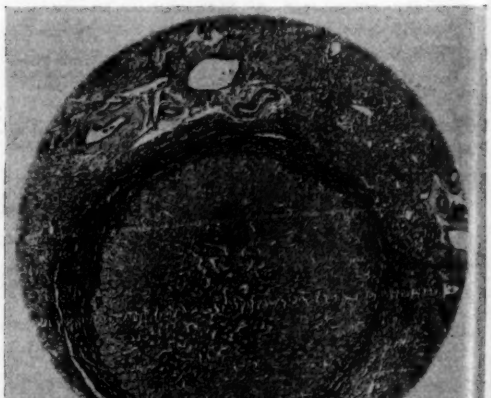


Fig. 10. Single adenomatoid nodule of hyperplastic liver cells, with great loss of hepatic substance around it.

at the ampulla of Vater was the cause of the condition. There had been a prolonged bile block for months. The icterus index averaged 60, there was no bile in the stool, and no urobilinogen in the urine. The van den Bergh was biphastic.

The liver has a remarkable power of regeneration. If not overwhelmed at first by the acuteness or the rapid progression of a disease, it will replace gradually, at least in part, the substance lost.

In conclusion, it can only be said that, in arriving at a clinical diagnosis in a case of jaundice, a careful preliminary survey of the presenting features, combined with a discriminating selection of the aids to a functional liver study, will be of assistance in a differential analysis—perhaps not indispensable, but at least helpful in the solution, and certainly of intriguing possibilities for a wider understanding of liver physiology and pathological states.

REFERENCES

Hepatitis, Deaver, J. B., J. A. M. A., 96:1293, Apr. 18, 1931.

Toxic effects following use of arsphenamine. Cole, H. N. et als, J. A. M. A., 97:897, Sept. 26, 1931.

Significance of urobilogen in urine as test for liver function. Wallace, G. B. and Diamond, J. S., Arch. Int. Med., 35:698, June 1925.

Diseases of the biliary tract. Dixon, C. F., Calif. & West Med., 35:1, July, 1931.

Interrelation of bile pigment and hemoglobin. Editorial, J. A. M. A. 96:1310, April 18, 1931.

Physiology, correlations and technic of the van den Bergh reaction, icterus index and quantitative serum bilirubin. Elton, Norman W., J. Lab. & Clin. Med., 71:, October, 1931.

Functional aspects of regenerated hepatic tissue. Althausen, T. L., Arch. Int. Med., 48:667, Oct., 1931.

Decompensated portal cirrhosis. Chapman, C. B., et als, J. A. M. A., 97:237, July 25, 1931.

Metabolism of galactose. Rowe, A. W. and McManus, M., Am. J. Med. Sc., 181:777, June, 1931.

Metabolism of galactose. Shay, H., et als, Arch. Int. Med., 47:391, March, 1931.

Galactose tolerance test in differential diagnosis of jaundice. Shay, H., et als, Arch. Int. Med., April, 1931.

A new liver function test. Delprat, G. D., et als, Arch. Int. Med., 34:533, Oct., 1924.

The rose bengal test for liver function. Kerr, W. J., et als, J. A. M. A. 85:942, Sept. 26, 1925.

The rose bengal test for liver function. Epstein, N. N. and Rauschkolb, J. E., Arch. Dermat. and Syph., 14:122, Aug., 1926.

The present status of liver function tests. Althausen, T. L., Calif. and West. Med., 32:53, Jan., 1930.

The rose bengal test for liver function. Delprat, G. D. and Stowe, W. P., J. Lab. & Clin. Med., 16:923, June, 1931.

Surgical Diagnosis (v. III), Graham.

NEPHROSIS

NELSON W. BARKER, M. D.,
and
EDWIN G. BANNICK, M. D.,
Section on Medicine, The Mayo Clinic,
Rochester, Minnesota

(Read before the Seventeenth Annual Meeting of the Medical and Surgical Association of the Southwest, Phoenix, Arizona, December 3 to 5, 1931.)

Much confusion and much disagreement have arisen with regard to the classification of diffuse renal lesions. In a given case the clinician is faced with the problem of expressing, in terms of renal pathologic anatomy only, a clinical syndrome which includes the results of laboratory tests, much of which may concern extrarenal factors. Hence has come the multiplicity of terms for similar diseases, and difference of opinion regarding the definition of terms commonly used. The exact etiology and pathogenesis of renal lesions and their extrarenal complications is still obscure but some progress has been made with regard to disturbed bodily functions and chemistry. At present, treatment is based much more on these latter factors than on the diagnosis of the renal lesion itself. However, for purposes of classification, the attempt is still made to adhere to existing and fairly well-accepted terms descriptive of pathologic anatomy, until more progress is made with regard to etiology.

In order to try to correlate clinical and anatomic findings, we studied clinical data and material obtained at necropsy in 200 cases of diffuse renal disease of various types. Our control material consisted of kidneys obtained at 250 postmortem examinations in which no clinical evidence of renal disease was obtained. This study has been supplemented by clinical data on 250 more cases. From these data we have attempted a classification similar, in a general way, to that of Volhard and Fahr. In such a classification cases of diffuse renal disease are divided into three main groups: nephrosis, in which lesions are of a degenerative type; nephritis, in which lesions are of an inflammatory and proliferative type; and nephrosclerosis, in which lesions are due primarily to vascular insufficiency and chronic ischemia. This paper is concerned with the first of these groups, and is based on our observations in the cases mentioned.

The term "nephrosis" has been criticized as ambiguous, since it simply means "disease of the kideny." It was first used by Friedrich Muller in 1905 to indicate a group of renal lesions of a degenerative type which he felt should be distinguished clinically and morphologically from inflammatory glomerulonephritis. Since then the term nephrosis

has been accepted by a number of students as applicable to all degenerative renal lesions. Many pathologists offer a more serious criticism, namely, that there is no sharp distinction between inflammation and degeneration of the kidney, and even that there is no degeneration without inflammation. Possibly this is a justifiable attack but it reduces almost all renal disease to degrees of nephritis and tends to more confusion. Also, typical nephritis or glomerulonephritis is characterized, even in acute cases, by definite exudation of foreign cells, and by proliferation of glomerular ephithelium and endothelium, and, in more longstanding cases by extensive scarring and destruction of all the functioning elements of the kidney. There is certainly a large number of cases of diffuse renal disease in which these evidences of inflammation are not seen.

We wish to define nephrosis, in its broad sense, as a diffuse renal lesion characterized by noninflammatory, nonproliferative, retrogressive, or degenerative changes in the parenchyma. The changes are usually uniform throughout but are of relatively mild degree. They are most easily observed in the epithelial cells of the convoluted tubules, but the epithelial cells of the glomeruli, and also the basement membrane, may be affected. Many types of degenerative or retrograde change have been described, such as albuminous, hyaline, hydropic, lipoid, amyloid, calcareous, and uric acid. In our experience a very common lesion, which may occur independently or in conjunction with these changes, is diffuse atrophy of the parenchyma, flattening of the epithelial cells of the tubules with maintenance of their definite outlines, and widening of the intertubular spaces due to edema or, in cases of long standing, to a deposit of loose fibrous tissue; usually, but not always, there is some cellular atrophy and shrinkage of the glomerular tuft also. Occasionally this parenchymatous atrophy becomes extreme; sometimes it is very slight. In fulminating cases there may be actual necrosis. It is often impossible to predict the exact type of degeneration from the clinical picture, but in these kidneys definite inflammatory changes are not found.

There are two main groups of nephrosis from the clinical standpoint, the acute and the chronic. The development and clinical pictures of these differ widely, but the anatomic renal lesions may be similar. One clinical characteristic of all types of nephrosis is absence of evidence of cardio-vascular injury, namely, hypertension, retinitis, arteriosclerosis, or cardiac hypertrophy.

ACUTE NEPHROSIS

Acute nephrosis is characterized in general by two outstanding clinical findings, oliguria and azotemia. Albuminuria and cylindruria are variable, not always striking. Hematuria is rare except in cases due to chemical poisoning. Edema is rare except as a late complication if the intake of fluid has been liberal.. Two groups, which can be distinguished by their etiology, are acute chemical nephrosis and acute toxic nephrosis.

Acute chemical nephrosis. The most frequent cause of this condition is ingestion of mercuric chloride. Other poisons which can produce it are uranium salts, chromium salts, arsenic, tartrates, phenol, and fluorides. It has affected patients who seemed to have an abnormal sensitiveness to arsphenamine.

The kidneys are usually large, pale, and tense, and in fulminating cases may contain multiple small hemorrhages. Histologically, particularly in severe cases of poisoning by mercury, the reaction is the most intense found in any of the forms of nephrosis; namely, actual necrosis of the tubular epithelium, with hemorrhages usually around or into the tubules. In other cases there may be only atrophy of the epithelial cells and intertubular edema.

The clinical picture is that of increasing azotemia, and elimination from the kidneys may range from oliguria to actual anuria. The small amount of urine may contain albumin, blood and casts. Cardiovascular changes and edema are very rare, but slight hypertension may occur as a late phenomenon in fatal cases.

Acute toxic nephrosis (acute toxic renal insufficiency). This is one of the most common types of diffuse renal disease. It occurs in the course of many cases of toxemia of high grade, particularly in peritonitis, jaundice, empyema, septicemia, toxic gangene, intestinal obstruction, both of the upper and lower parts of the bowel, pneumonia, malaria, and (rarely) after intravenous administration of foreign protein. The renal changes are not consistent. Occasionally there is little histologic change in the kidneys. Degeneration of the hyaline droplet, hydropic, or lipoid types are occasionally observed. The kidneys are frequently enlarged, pale, and tense, sometimes they are small, soft, and friable. Hemorrhages are unusual. In the majority of cases there is diffuse and definite, but not extreme, atrophy of the tubular epithelium, with some shrinkage of the glomerular tufts, and slight but definite intertubular edema.

The clinical observations in acute toxic nephrosis are oliguria and azotemia. Albuminuria is slight to moderate, if present at all. Hematuria and cardiovascular changes

are rare as distinguished from acute glomerulonephritis.

The acute toxic nephrosis of jaundice is perhaps worthy of special mention. We have seen a number of these cases occurring in the presence of cholemic states, particularly in association with the various types of obstructive jaundice. Exactly what precipitates renal failure is not known, for the latter does not necessarily parallel the degree of jaundice. It is sometimes precipitated by a surgical procedure applied to a jaundiced patient. Many of the patients are elderly and somewhat cachectic, and the kidneys of many of them have undergone previous arteriosclerotic injury. At necropsy, the kidneys are not often found to be enlarged, are usually pale greenish-brown, and soft. Histologically diffuse tubular atrophy and intertubular edema are almost constant observations, and the tubular cells usually contain bile pigment.

Treatment. Treatment of all forms of acute nephrosis follows the same plan. In the toxic conditions mentioned, early recognition of renal insufficiency is important, and necessitates careful daily records of intake and output of fluid. The cause of the toxic condition itself is usually apparent. In cases of chemical poisoning, as much as possible of the poison should be removed from the gastro-intestinal tract, and efforts should be made to neutralize the remainder. In toxic nephrosis, every effort should be made to treat the underlying disease. For the renal condition itself, a diet low in protein and salt (40 Gm. of protein a day) and a high intake of fluid (up to 3,000 cc. a day) are essential. This amount of fluid may have to be reduced if the oliguria persists. If intravenous administration of this fluid is necessary, a solution of 5 to 10 per cent glucose is preferable to physiologic solution of sodium chloride; sodium chloride is not well excreted by these kidneys and may accumulate in the body enough to cause edema, in which case treatment is considerably complicated. An exception to this is the acute nephrosis of obstruction high in the intestinal tract, in which the value for chlorides in the blood is low and administration of physiologic solution of sodium chloride is indicated. The caffeine group of diuretics may aid materially in increasing the urinary output. In chemical nephrosis with anuria, a few brilliant results have followed decapsulation. If anuria persists, decapsulation should be considered. A beneficial effect may result, if at operation the kidneys are found to be large, swollen, and congested.

The prognosis in acute toxic nephrosis is good if the underlying cause can be removed. Normal renal function can be reestablished, and anatomically minimal permanent organic injury would be expected.

CHRONIC LIPOID NEPHROSIS

This condition has been well established as a definite clinical syndrome. It seems to affect persons of all ages, but particularly children and young adults. The outstanding observations are generalized edema and albuminuria of high grade; casts may or may not be present. Hematuria, cardiovascular changes, and anemia do not occur. Azotemia is absent in typical cases; whether it ever occurs, even as a late phenomenon, is debated, and its presence should raise a question as to the diagnosis. Lowering of the value for serum protein, with inversion of the albumin-globulin ratio, and increase in the value for blood cholesterol, are almost always found. It is important to remember that all the manifestations mentioned may be found in certain stages of subacute glomerulonephritis, but with the addition of varying degrees of hematuria, azotemia, and cardiovascular change, including hypertension. In chronic lipoid nephrosis the basal metabolic rate may be slightly subnormal. There is rather definitely increased susceptibility to infection. The etiology and pathogenesis of chronic lipoid nephrosis are still debated. One theory, proposed by Epstein and his coworkers, is that the condition is primarily a metabolic disease, in which a toxic protein is formed, which the kidney secretes as it would secrete any foreign protein, and that the kidney itself is little affected except secondarily. A second theory, which Bell advocated, is that primarily a true form of inflammatory glomerulonephritis exists, which undergoes partial healing, but which leaves an injured kidney, with abnormally permeable glomeruli and partly degenerated tubules. A third theory, upheld by Fahr and others, is that the condition is a primary, idiopathic, degenerative renal disease which affects the glomerular epithelium as well as the tubules, and renders the former permeable to serum albumin. It is possible that clinically indistinguishable cases of the syndrome of lipoid nephrosis may be produced in all three ways. In certain cases, the clinical picture of glomerulonephritis subsequently develops and, when patients die, lesions of glomerulonephritis are found. Hence, a given patient should be observed several months before a diagnosis of uncomplicated lipoid nephrosis is made. However, such uncomplicated cases do occur, and as distinguished from chronic glomerulonephritis, the prognosis is good, particularly if treatment is carried out carefully. We have

seen a number of cases in which all symptoms and abnormal clinical findings have disappeared. In atypical cases, or cases of mixed lipoid nephrosis and glomerulonephritis, the less the evidence of nephritis (hematuria, cardiovascular changes, azotemia, and anemia), the better the prognosis.

Not many observations at necropsy in cases of uncomplicated chronic lipoid nephrosis have been reported, for the patients do not die of renal failure. The condition may be chronic, and may persist for a number of years, with exacerbations and remissions. When the patients die, the terminal picture is usually that of edema of the brain, cardiac collapse, and cellulitis of edematous regions, or of infection such as pneumonia, peritonitis, or septicemia. Some cases have been reported in which there were few changes in the kidneys. In our cases, the kidneys have been pale or yellowish, and usually enlarged. The histologic picture is usually that of diffuse, rather uniform atrophy of the epithelial cells of the tubules and glomerular tufts, with or without deposits of fat. Special strains frequently disclose some thickening of the glomerular basement membrane. Fahr described an advanced stage, the so-called nephrotic, contracted kidney.

Treatment is based on the clinical observations, which differ strikingly from those of acute nephrosis, and is particularly directed toward relief of edema. This has been considered previously by Bannick and Keith. With the absence of azotemia, many clinicians feel that a diet high in protein (70 to 100 Gm. daily) is desirable, if the patient tolerates it, in order to compensate for the drain of albumin through the kidneys, and for the low value for serum protein, which probably is a factor in production of the edema. The intake of sodium chloride should be decreased to an absolute minimum and the intake of fluids decreased to from 600 to 800 cc. daily. Diuretics of the caffeine group, those of the acid-producing salt type, and mercurial diuretics frequently accomplish brilliant results in relief of edema. Thyroid extract seems to help in a few cases. Care should be taken to avoid respiratory and other intercurrent infections.

AMYLOID NEPHROSIS

The kidney is one of the three organs in which amyloid is most commonly deposited. The most common cause of amyloidosis are chronic infections such as tuberculosis, osteomyelitis, syphilis, actinomycosis, and lymphoblastoma. In other cases the causative factors may be very obscure. In the kidneys, amyloid becomes deposited chiefly in the glomeruli and walls of the blood vessels. Occasionally it is found around the tubules. In advanced cases, varying degrees of secondary tubular degeneration occur, which resemble very much the atrophy found in other types of nephrosis. Grossly, the kidneys are usually large and pale.

Renal amyloidosis may be found at necropsy in cases in which there was no clinical evidence of renal disease. Cases of amyloid disease are described in which the clinical picture has resembled that of lipoid nephrosis, but more commonly there is albuminuria, slight hematuria, and slight azotemia, lowering of excretion of phenolsulphonphthalein and, particularly, inability of the kidneys to produce normally concentrated urine, without edema. Casts may or may not be found in the urine; waxy casts are supposed to be pathognomonic. When amyloidosis is suspected, the Congo-red test usually will settle the diagnosis. This test was first described by Bennhold. At the Mayo Clinic the technic has been to give, intravenously, 18 cc. of 1.5 per cent fresh solution of Congo red in triply distilled water, and compare concentration of dye in the blood serum withdrawn one hour later with that of serum withdrawn four minutes after injection of the dye. Any considerable amount of amyloid in the body will cause disappearance of at least 75 per cent of the dye from the blood stream in this period. Little is known of the prognosis and course of amyloid disease, for relatively few cases have been diagnosed clinically. Since the advent of the use of the Congo-red test, the diagnosis has been made more frequently than before, and data as to prognosis may be forthcoming. The chronic infection or systemic disease is usually of primary importance, and the renal disease is insufficient to explain the patient's death. If renal insufficiency without edema is present, intake of protein should be restricted and intake of fluid increased.

Finally, there are a number of cases of albuminuria without edema, demonstrable renal insufficiency, or evidence of cardiovascular injury, in which classification and prognosis are difficult. In some of these cases the albuminuria is undoubtedly residual from previous glomerulonephritis, or indicates a low grade of inflammatory lesion. The history, and careful studies of renal function are important in this group. Other cases apparently represent a mild degenerative process; hence, they may be classified as a type of chronic nephrosis, and eventually the value for serum proteins may be lowered, and even edema may appear. Observations at necropsy, in a few cases in which unrelated diseases have caused death of the

patients, have failed to disclose evidence of glomerular inflammatory change.

SUMMARY

If the term "nephrosis" is accepted to designate a group of diffuse renal lesions of a mild but definitely retrogressive or degenerative nature, without evidence of exudation, proliferation, or arterial insufficiency, there are four clinical groups which may be placed in this category. Of these, chemical nephrosis and toxic nephrosis are acute lesions characterized by oliguria and azotemia. Chronic lipoid nephrosis is clinically a very different entity, characterized by edema and albuminuria of high grade, without hematuria, cardiovascular changes, or azotemia. Amyloid disease of the kidneys may be diagnosed clinically by the Congo-red test; in this condition clinical symptoms of renal disease may be absent, or there may be mild renal insufficiency and mild hematuria, with or without edema. The treatment of all these conditions is dependent on removal of the cause, in cases in which it is known, and on the disturbed physiology and physiologic chemistry, not on the nature of the renal lesion itself.

BIBLIOGRAPHY

1. Bannick, E. G.; and Keith, N. M.: The treatment of nephritis and nephrosis with edema. J. A. M. A. 91:1944-1952 (Dec. 22) 1928.

2. Barker, N. W.; and Snell, A. M.: The Congo-red test with special reference to excretion of the dye in the urine. Jour. Lab. and Clin. Med. 16:262-270 (Dec.) 1930.

3. Bell, E. T.: A text-book of pathology. Philadelphia, Lea and Febiger, 1930, 627 pp.

4. Bennhold, Hermann: Uber die Ausscheidung intravenos einverleibten Kongorotes bei den verschiedensten Erkrankungen insbesondere bei Amyloidosis. Deutsch. Arch. f. klin. Med. 142:32-46 (March) 1923.

5. Epstein, A. A.: Futher observations on the nature and treatment of chronic nephrosis. Am. Jour. Med. So. 63:167-186 (Feb.) 1922.

6. Fahr, T.: Pathologische Anatomie des Morbus Brightii: In: Henke, F., and Lubarsch, O.: Handbuch der speziellen Pathologischen Anatomie and Histologie. Berlin, Julius Springer, pt. 1, 6:156-472, 192g.

7. Volhard, F.: Die doppelseitigen hamatogenen Nierenerkrankungen (Bright'sche Krankheit). Berlin Julius Springer, 1918, 576 pp.

CARCINOMA OF THE PROSTATE

DAVID M. DAVIS, M. D.
Phoenix, Arizona

(Read before the Seventeenth Annual Meeting of the Medical and Surgical Association of the Southwest, held at Phoenix, Ariz., Dec. 3-5, 1931.)

A necessary preliminary to the treatment of carcinoma is the proper diagnosis. Since uncomplicated carcinoma of the prostate usually causes no symptoms for a long time, the average case is well advanced when first seen by a physician, and there can be little doubt about the diagnosis. It is incorrect, however, to assume that diagnostic errors do not occur in this disease. They are of two kinds: first, missing a carcinoma; and, second, mistaking another condition for carcinoma. The first mistake is by far the commoner.

In a series of almost two thousand cases of operation for prostatic obstruction, the tissue removed at the operation showed carcinoma in 250 cases. I studied the pathological material and clinical records of these 250 cases, and found that in twenty-five the carcinoma had not been recognized before operation. The reasons for failure to make the diagnosis can be divided into four general groups, as shown in Chart I.*

Group I. In seven cases the tumor, while a typical prostatic adenocarcinoma microscopically, had so little stroma that it lacked the hardness usually seen. Five of these escaped diagnosis entirely until they came under the microscope. We may say, then, that in this series 2.8 per cent of the carcinomas were soft, and not recognizable by the usual criteria upon rectal examination.

Group II. In five cases the prostates were indurated, but did not have the stony hardness usually found, and the examiners thought of chronic inflammatory changes rather than malignancy.

Group III. In five cases a hard nodule was felt, but its smallness deceived the examiners.

Group IV. In eight cases the carcinoma was obscured by a coexistent benign hypertrophy. In four a small nodule of carcinoma was entirely surrounded by elastic hypertrophied tissue. In one, the tumor grew in a thin layer in the posterior lamella, and was not perceptible, owing to the elasticity imparted by the mass of hypertrophic tissue beneath. In four, the tumor grew as a hard

Error due to →	I Soft type of tumor	II Error in judging induration.	III Small size of nodule	IVa Surrounded by B.P.H.	b Thin layer over B.P.H.	c Nodule over B.P.H.	Total %
Diagnosed at operation, suspected before.	1	2	1	0	0	2	6
Diagnosed at operation, unsuspected before.	1	0	0	0	1	1	3
Diagnosed by microscope only.	5	3	4	4	0	0	16
	7	5	5	4	1	3	25

*This table has been published in Young's Practice of Urology, by Hugh H. Young and David M. Davis.

nodule in the posterior lamella, but had a sense of elasticity imparted to it in the same way by the hypertrophied tissue beneath.

It should be noted that this series of undiagnosed cases is 10 per cent of the cancer cases, and about 1 per cent of all the operations for prostatic hypertrophy, and cannot therefore be dismissed as entirely negligible. In nineteen cases, or 7.5 per cent of the cancer cases, there was not even a suspicion of the presence of carcinoma. Sixteen of the cases were not diagnosed, even at the operating table. This demands that frozen sections be made in every case where anything in the least suspicious is found.

As a result of this study, the following suggestions are made in the hope of diminishing the number of missed diagnoses:

1. Extensive induration with adhesions and fixation of the prostate, especially if involving the seminal vesicles, should always cause the examiner to suspect carcinoma, even if the induration is not stony.

2. Nodules markedly finer than the surrounding prostate suggest the possibility of carcinoma. Spheroids of hypertrophy may occur in the posterior lamella, but are so rare that such nodules should suggest carcinoma.

3. Thickening of the membranous urethra, fixation, and the obscuring of its outlines, suggest the inflammatory reaction occurring with carcinoma.

4 The same is true of similar changes in the notch between the two seminal vesicles.

5. Thickening and induration of the lymphatics and lymph nodes about the tip of the seminal vesicle suggests the possibility of carcinoma.

6. Examination with the finger in the rectum and a metal instrument in the urethra enables one to appreciate better the degree of induration present in the posterior lamella.

7. The presence of benign hypertrophy, shown by rectal or cystoscopic examination, in no way excludes carcinoma.

The three diseases most commonly mistaken for carcinoma of the prostate are tuberculosis, chronic prostatitis, and prostatic calculi.

The prostate of tuberculosis may be exactly like that of cancer, as far as rectal examination goes, but other lesions in the epididymis, bladder, or kidney, are usually apparent. In the rare cases when these are absent, and the patient is of the cancer age, errors may be made. These errors are serious, since operation on a tuberculous prostate is apt to lead to permanent fistula. Careful study of the urine and prostatic secretion can prevent most of them.

The induration of chronic prostatitis distorts the prostate less and is less extreme than that of carcinoma, yet it may cause errors. I have seen two cases in which positive diagnosis of carcinoma was made, and a third in which it would probably have been made had not the patient been a young man in his twenties.

Prostatic calculi should give no trouble unless one forgets to take an x-ray.

Syphilis and actinomycosis might simulate carcinoma, but are very rare.

I believe that carcinoma of the prostate should always be treated, but the type of treatment to be adopted depends on the stage of the disease. There are only four kinds of treatment; namely: radical operation for complete removal, conservative operation for the relief of obstruction, radium, and x-ray.

Radical removal should be carried out in all cases, with or without symptoms of obstruction, in which the growth is entirely confined to the prostate and in which there is no evidence of metastasis. Unfortunately, one sees very few of these early cases, owing to the insidious course of the disease. For removal the method of Young is to be preferred. This operation is done through the perineum, and in it the entire prostate, prostatic urethra, seminal vesicles, and a portion of the bladder immediately surrounding the vesical orifice, are removed. There is risk that urinary control will not be perfect after the operation, but since there is no other way in which we can even hope for a real cure, one is justified in taking this risk. Coffey suggests that the bladder also be removed, with transplantation of the ureters into the sigmoid. Since early carcinoma so seldom involves the bladder, it would seem inadvisable in most cases to make the operation so much more extensive and dangerous.

If the case is unsuitable for radical operation, radium should be used. My own preference is for the platinum or gold-coated emanation seeds, implanted by needles inserted through the perineum. Occasionally one may implant part of the seeds through an endoscope, as the median and subtrigonal regions can be reached better in this way, or through a perineal incision. With this method, doses as large as desired can be inserted at one sitting without discomfort to the patient and without danger of rectal or urethral irritation or ulceration. Studies at the Memorial Hospital in New York indicate that very large doses can be used in this manner, and that better results are obtained with them. Martin, Quimby, and Pack have evolved a method of calculating the amount of radium necessary for successful treat-

ment of various types of tumors. Since the carcinomatous prostate is often of good size, it is easy, unless one is familiar with this work, to give insufficient doses. Some cases, particularly those with the softer type of growth, show little or no response to radium, but in many others striking symptomatic improvement occurs, and the prostate shows definite softening and reduction in size. In a recent case the patient, who formerly had a nocturnal frequency of seven or eight times, now sleeps through the entire night. In short, the chances of benefit from radium are so great and the danger of untoward effects so slight, that I do not believe it should be withheld from any patient. The two things to avoid are: first, too small doses, which may stimulate the growth; and, second, insertion of a seed or needle too far, so that it passes beyond the seminal vesicle to a point where it may set up a retrovesical infection. This latter event is, fortunately, very rare.

Even if there is well-marked urinary obstruction, one may await the results of radium treatment unless the obstruction is severe enough to cause great misery or to interfere greatly with renal function. In this event the nature of the obstruction must be discovered by cystoscopy. If it is of suitable size and location it can be removed by a transurethral procedure such as a punch operation, the electrotome method of Collings, or the cutting-loop method of Stern, as modified by T. M. Davis or MacCarthy. If this is not possible, a channel through the prostate can be made by operative removal of the central portion of the growth. This method is greatly preferable to the creation of a permanent suprapubic fistula, which should be reserved for the most debilitated and hopeless cases. The conservative operation can be done by the suprapubic approach, but the perineal method is much more satisfactory. If the prostate is large and entirely infiltrated with a scirrhous type of growth, this procedure may be extremely difficult by either route. I have experimented with the electric scalpel and special types of cutting instruments, and hope soon to be able to suggest a method for meeting the requirements of this situation in a manner which will reduce trauma to a minimum and at the same time be entirely safe.

I have never seen any important reason for not placing radium emanation seeds in the remaining portions of the prostate and seminal vesicles after conservative operation on the carcinomatous prostate. Healing does not appear to be delayed, and certainly the seeds can be placed more accurately at this time than at any other.

X-ray appears to have little effect on the prostate itself in carcinoma. Deep therapy, however, is strikingly effective in relieving the pain due to metastasis, particularly the common lumbago and sciatica, and is a real godsend in this respect. X-ray applied directly to the prostate, in combination with radium treatment, may have some synergistic effect. There is little real evidence to support this idea, but it may be tried in a situation where neither doctor nor patient wishes to leave any stone unturned which may offer even a remote chance of benefit.

In far-advanced cases with invasion of the bladder, rectal ulceration, or rectal obstruction, the treatment must be purely palliative. Extension to the bladder may demand suprapubic cystostomy. In such cases urethral involvement may usually be relied upon to bring merciful termination, and nephrostomy or ureterostomy is seldom justified. For rectal irritation, suppositories or nupercaine, or even cocaine, are very useful. Rectal obstruction and intense pain resulting therefrom may necessitate colostomy. When pain alone is the dominant factor, cordotomy is the logical procedure and should be done if it appears that the patient will survive more than two or three months.

The patient with carcinoma of the prostate is sometimes abandoned to his fate, and this, I think, is unjustifiable. It is the duty of the physician to relieve as well as to cure, and I have tried to show that he has many effective methods at his command in this disease.

GENERAL CONSIDERATION IN THE HANDLING OF FRACTURES, TAKING A FRACTURE OF THE FEMUR AS AN EXAMPLE

W. S. SHARP, M. D.
McNary, Arizona

(Read before the Seventeenth Annual Meeting of the Medical and Surgical Association of the Southwest, held at Phoenix, Ariz., Dec. 3 to5, 1932)

I am speaking from the standpoint of the industrial surgeon, upon whose shoulders fractures weigh most heavily. I am speaking too, from the standpoint of one whose cases occur in the most unfavorable surroundings. The patient will be a man who probably has not had a bath in weeks, whose clothing is not changed or washed often, who is usually covered with grease, grime, dust and everything most unfavorable to a lacerated wound of any degree, to say nothing of a protruding fragment of bone that has been drawn back into the interior of a limb. These

are quite different from the fractures seen in clean individuals and that occur in better surroundings.

In the city where an accident occurs a warm comfortable ambulance is present within a very few minutes. With the corporation whom I am serving, our most serious accidents occur at the outposts, from fallen trees, fallen logs and timber, wrecked trains, and so forth, sometimes ten or fifteen miles from the hospital, with deep snows in winter, and heavy rains in summer, often preventing other transportation than by log train. I am not familiar with the situation in the gold, silver and copper mining centers, but am confident that you have your problems along similar lines.

Within the last few years greater interest has been shown by the profession everywhere in the treatment of fractures, in the fracture equipment of hospitals, and in the qualifications of the men doing fracture work. This has been brought about to a great extent by recognition of the inadequate treatment of fractures and uncertain results obtained. Also by the activity of the American College of Surgeons whose committee, headed by Dr. Charles L. Scudder, has set about to standardize as nearly as possible the treatment of fractures, to improve hospital facilities, and to impress upon the surgeons the inadequacy of old and indirect methods of traction. This committee, which has laid down rules for the adequate equipment of hospitals, has also submitted requirements for surgeons doing this work, which I will enumerate.

(1) The procedure must be with the highest degree of safety to the patient.

(2) Surgeons and assistants should be skilled in this treatment.

(3) They should have proper instruments and appliances for their work.

(4) The anesthetist should be skillful.

(5) Pre-operative, operative and postoperative treatment should be adequate.

(6) Method chosen should be the one indicated in the particular case.

(7) Contiguous joints should be moved as little as possible.

(8) The surgeon is held responsible for only the average in his community. He feels more should be given.

GENERAL CONSIDERATION IN ALL FRACTURES

(a) Avoid unnecessary handling or moving.

(b) Keep the injured man warm.

(c) Leave the limb or strap the member in the position it is found.

(d) In compound fractures, the clothing should be cut away, and if possible gauze or some clean dressing should be placed over open wounds or bone ends.

(e) Ease the man's pain.

At the plant where I am employed, each locomotive, steam loader, steel gang car, and major operating unit, such as sawmill, planer, machine shop, or power house, is supplied with a long box containing the following:

A two-man stretcher; two blankets; roll of heavy canvas six feet by ten feet; a small package containing sterile gauze, some bandages, and a smaller box containing two quarter-grain morphine tablets.

The foreman at monthly meetings are given first aid instructions. They are instructed to lay the stretcher down, and on top of that, the canvas and blanket. The injured man is placed on this, and the injured limb is strapped or bound by the bandages to the well limb, if such exists. The canvas is then folded and bound around the entire body and limbs. The canvas serves as a protection from rain or cold, and the man thus bound may be picked up bodily; he is thus transported directly to the x-ray room, and treatment then decided upon. If he is in a state of shock, this is combatted. All clothing is cut away. No attempt is made to remove clothing intact. It is a routine measure to administer by hypodermic the prophylactic dose of tetanus antitoxin. He is given ease from pain, and kept as warm as possible. The fracture is reduced as early as safety to the patient and circumstances will permit. At all times gentleness in handling and a minimum of handling is insisted upon.

We are indebted to Dr. Scudder's committee, from the American College of Surgeons, for the following recommendations as to the equipment of hospitals: The hospital should be well supplied with adequate and approved fracture appliances, especially sheet wadding and rolls and sheets, plaster of Paris, various metallic splints and their modification, overhead frame or "grape arbors" for overhead suspension; screw-pullers may be placed in the frame anywhere, small strong rope, weights and a fracture bed, a Hawley table and an x-ray plant. In addition to this, for open reduction of fractures, metal plates of various lengths, metal bands, wire, kangaroo tendon, bone-holding forceps, and so forth, should be at hand. Also material for skeletal traction.

So many hospitals have neglected these details and, as a result, fractures have been difficult to handle and their proper reduction delayed.

Fractures of the neck of the femur are seen most frequently in the aged, and require the services of one who gives special attention to this work. Fractures of the upper third of the shaft are important because of the difficulty of reduction and the frequent deformity that follows. Fractures in the middle third of the shaft may occur in various forms, such as oblique, with marked overriding, or transverse. The chief characteristic from the standpoint of end result is the marked shortening that may result from an unreduced fracture. In addition to the marked shortening, angulation deformity is not uncommon. Fractures of the lower third of the femur are important because of the frequency with which they involve the knee joint and its associated structure, resulting in shortening and such deformities as knock knee, restricted motion, instability, effusion and pain. Displacement of fragments in fracture of shaft of the femur is far greater than in fracture of any other bone on account of the length of the femur, and the powerful muscles attached. In transverse or even slightly oblique fractures, where separation is complete, the fragments override at once, and in very oblique fracture, the sharp point of the upper fragment may penetrate the muscles and appear at the skin surface, although indirect violence may be the cause.

Usually the distal fragment is drawn upward and behind the proximal one, on account of the muscular contraction and infiltration of the thigh, and an angularity directed outward and forward occurs. The lower fragment rotates outward, turned by the weight of the foot and leg, and rarely turns inward.

In upper third fractures the proximal fragment is, as a rule, pulled forward and outward by the muscles attached to the trochanters, the gluteal muscles and the psoas muscles, while the flexors and adductors draw the lower fragment inward and up against the upper fragment.

If the periosteum remains intact in one part of a fracture, holding the fragment ends together, the shortening results from the angularity of the fragments, but if the periosteum is stripped off either fragment sufficient to allow a slipping by, or is torn completely, shortening in addition to angularity is caused by the overriding. Some rotation is present in all cases.

The most serious complication in any fracture is fat embolism. This complication is more liable to occur in fracture of the shaft of the femur, or that portion of the femur from the lesser trochanter to the condyles; first, because of the unusual displacement of the fragments brought about by the divergent pull of the muscles upon the upper and lower fragments; second, because of the proximity of the larger vessels; assuming that the theory of absorption of the medullary substance of the bone by the return flow of blood to be correct, and I have heard of no more plausible theory. The two cases of this complication occurring in my practice have both been fatal, and in both instances the break has occurred in the middle and upper thirds of the bone. Fractures of the upper and lower thirds of the femur, which are likely to resist forcible traction and reposition, should be replaced by operation. Certain transverse fractures whose fragments can not be approximated because of muscle interposition should be operated upon. Transverse fractures having interposing fragments of bone should be operated upon. Long oblique and oblique fractures that defy traction should be openly reduced.

The limitations of traction and countertraction, of manipulation and pressure, together with suspension, as the adequate treatment of fractures of the shaft of the femur are gradually being recognized. As these limitations are better known, much unnecessary experimental manipulative treatment will be abandoned.

The successful and adequate method of treating a given fracture should be selected at the outset. There will always be fractures offering varying difficult problems in their management. There will always be variations from normal anatomical and functional results in any non-operative or operative treatment. These facts make it incumbent on the operator to give each case special study.

In fractures of the femur where adequate fixation, such as a Lane plate, has been used, such authorities as Scudder and Sherman do not use the plaster cast, but employ the Thomas splint with overhead suspension. Circumstances will alter this routine. For instance, fracture of the upper third of the femur, where there is comminution and difficulty in attaching a Lane plate to the fragments, a spica cast would be preferable.

In fractures of the femur, more than anywhere else in the body, we have diverging muscular pull on the upper and lower fragments to deal with. In these cases, and especially where there is much overriding or separation, or comminution in the fragments and where there is doubt as to the interposing of muscle and bone fragments, open

reduction should be resorted to as early as possible. The longer this procedure is delayed, the greater the difficulty from contraction of muscle and fascia, thus requiring additional manipulation and handling and affording a greater opportunity of contamination with its disastrous results.

In open reduction, especially where there is damage to the surrounding soft parts, and where debridgement and manipulation are necessary, I think a rubber tissue drain for forty-eight hours is a distinct advantage. I know this will not meet with the approval of several present, but experience has proved it to be a wonderful safety valve.

PROGNOSIS IN FRACTURES OF SHAFT OF THE FEMUR

The prognosis in fractures of the shaft is of interest in three main directions; mortality, functional results, and cosmetic results. First to be considered is danger to life, which is most important, and is a serious question in open fractures especially. The fractures of the shaft are accompanied by much violence, shock and hemorrhage, and this factor takes first place in the prognosis. Next to be considered are good functional results. End to end or anatomical approximations are not absolutely necesasry for the attainment of good functional results, but to-day that is not so accepted with the benefits of skeletal traction so easily obtained. Finally, satisfactory cosmetic results must be considered; that is, good end to end reposition of the fragments without deviation of the angle of support.

This is the ideal result to be sought. Normal length is maintained and the patient does not have to learn new balance and habits or new muscular tricks to get about. The proper use of skeletal traction will avoid shortening and any deviation from the normal lines of weight bearing. At the plant where I am employed, a complete physical examination is required before an applicant is employed. Hardly a week passes that we do not see the bad results of the old indirect extension methods, such as shortening, angulation, ankylosis, and so forth.

SUMMARY

Handle with the idea of minimizing shock. Relieve pain and keep the injured man warm. Support the limb in the position it is found. Transport with a minimum of handling. Depend upon your x-ray findings. Decide upon your treatment and if patient's condition is favorable, avoid unnecessary delay.

THE CHANGING POLLEN PICTURE

E. W. PHILLIPS, M. D., F. A. C. P.
Phoenix, Arizona

(Read before Maricopa County Medical Society at Phoenix, Arizona, Jan. 25, 1932.)

Last spring an indignant botanist walked into the office bearing an armful of pollinating plants. The botanist was Jesse Knapp of North Hollywood, and his indignation arose from the fact that none of these potential hay-fever producers has been reported from Arizona. Most of his specimens had been gathered on the desert between Wickenburg and Marinette, and their occurrence there interests the scientist more than the clinician. If the few hardy souls who inhabit that region suffer from hay-fever they do not trouble physicians about it. But two of the plants came from the Salt River Valley close to Phoenix, and the sight of them provoked certain twinges of conscience, and led to an examination of specimens and records. Both plants were desert ragweeds: Franseria dumosa, the common name of which is supposed to be bush sandbur; and Franseria ambrosioides, the canyon ragweed.

Knapp's specimen of Franseria dumosa had been collected near the packing plant, and a limited quantity of it was found there growing alongside the common Franseria deltoides. This distribution continues along the Salt River banks as far east as I have had time to go; it is found also in irregular patches along the Gila, and a narrow zone of it runs across the base of the Salt River mountains. Among my pollens I found a specimen of dumosa pollen collected in 1923. The files yielded up an old letter from Mr. Robert Peebles, the Government botanist at Sacaton, in which he enclosed a specimen of its staminate flowers, described the plant and wondered whether it might not be causing hay-fever among his neighbors. His question seemed to be answered in the negative. At that time I was treating several patients from Sacaton, and found it easy to control their Franseria symptoms by administering an extract of the pollen of rabbit bush. So, that hay-fever plant had been neglected.

The other specimen was an old friend, Franseria ambrosioides, the canyon ragweed. It has a wide but thin distribution all over Central Arizona—thin, because the plant requires a fair supply of water. It cannot live on the open desert but must keep to the canyons and washes. In wet seasons it grows rank and pollinates profusely. In the spring of 1924, having used up my small supply of rabbit bush pollen, I substituted canyon ragweed pollen in the treatment of patients

who were having hay-fever from the rabbit bush, and it worked quite as well. It seemed to be of little importance, because, in quantity it is insignificant compared to the rabbit bush. But that was in the cycle of dry years. If the tree-ring record of Dr. Douglas holds true, we shall have several more wet years,

Fig. 1. View of so-called "sage-brush" on the desert, this being rabbit bush (Franseria deltoides), a closeup of which is shown in Fig. 2.

and in that event the canyon ragweed will greatly increase. It may be as well at this point to present a brief description of the three spring-pollinating desert Franserias, which might be called

THE DESERT RAGWEEDS

The commonest, the hardiest, and by far the most abundant, is Franseria deltoides,

Fig. 2. A close-up view of rabbit bush, (Franseria deltoides), incorrectly called "sage-brush."

the rabbit bush. Locally, and quite incorrectly, it is called sage brush (Fig. 1). It is a low sprawling bush (Fig. 2) with triangular gray-green leaves that have a sage-like smell. It is strictly an opportunist and it takes advantage of the spring rain, if there is any, to bloom inconspicuously at any time from the tenth of March to the first of May. Given a little moisture, it can release a great quantity of pollen, though the yield from each bush is small.

Next in order is the bush sandbur, Franseria dumosa (Fig. 3), so called from its fruit; though the fruit of the rabbit bush is also a bur. Dumosa, as has been said, has a spotty distribution in this region. It has a denser growth than the rabbit bush, its leaf is smaller and it pollinates profusely at the same season.

Fig. 3. A close-up view of the bush sandbur (Franseria dumosa).

The canyon ragweed, Franseria ambrosioides, has been described in part, (Fig. 4). It is taller than either of its congeners, distinctly a greener and livelier plant. It blooms a week or ten days later than the others and yields considerable pollen. It seems to be extending its range, and can be found along canal banks and in fence corners at the outer border of the irrigated valley.

Fig. 4. The canyon ragweed (Franseria ambrosioides), showing its more luxuriant development, as it grows only where there is plenty of moisture.

As related above, I had used canyon ragweed pollen. I lost no time in trying out that of the bush sandbar. I had at the time (April 1931) some thirty-three patients under treatment with rabbit bush pollen, and I tested all of them with an extract of dumosa pollen. In twenty-eight cases, the two pollens produced equal, or nearly equal, reactions by the intradermal method, with rabbit bush having a shade the advantage. In five cases, there was a pronounced difference in favor of the sandbur, and these cases are worth a brief description. One was a man, an old pollen asthmatic, who lived near Tempe and who was sensitive to everything in the ragweed group. He had first come under treatment in 1927, receiving in that year intensive treatment with rabbit bush pollen. His relief extended over the next season, but he returned in 1929 and 1930, obtaining only partial relief. He was not doing any better in 1931; in spite of full dosage he had an occasional sharp recurrence of his symptoms. The test dose of bush sandbur pollen excited a strong local reaction, and he had no further trouble. The treatment was, of course, continued through the season. The second patient was a woman, living near Tempe; her case resembled the first, and her response was equally satisfactory. The third was a railroad man on the Parker run, and his symptoms were worst at that end. He was relieved by the sandbur but not by the rabbit bush pollen, though on each trip he traversed a desert covered with rabbit bush. There must be considerable Franseria dumosa near the Colorado River. The other two patients were physicians; they were multiply sensitized, they live in Phoenix and they knew that a new pollen was being used. Both reported better results when dumosa pollen was included in their dosage.

Of the other twenty-eight patients who reacted to the two Franserias alike or nearly so, nineteen were treated with rabbit bush pollen only, and in nine cases the sandbur was alternated with rabbit bush. It made no difference; the tolerance was the same, and all twenty-eight got good results. Apparently the biologic relation of the spring franserias is very close; all three are present in the air at the same time and group sensitization is the rule, specific sensitization the exception. A combination of the franseria pollens in the proportion of, say, seven parts of deltoides, two parts of dumosa and one of ambrosioides would approximate the distribution and it would probably take care of the occasional minority reactor. I shall try it next spring and report the result.

RUSSIAN THISTLE

Five years ago I ventured to make this prediction[1]: "I have watched the Bermuda grass creeping up along the watercourses to higher and cooler climatic zones, until now it is well established at 3500 feet and is beginning to take hold in some places at an altitude of 5000 feet or higher. The Russian thistle, traveling after the manner of tumbleweeds along roads and rights of way, has been adapting itself to lower and hotter zones until now there are small patches of it scattered all over the Valley. Growing population, greater acreage in use, more irrigation, more travel, more shipments of weed-fouled fodder, are the factors that are combining to erase our natural botanical boundaries. So it is probable that within a few years we shall have to deal with a more complex hay-fever situation in Arizona and we need to be alert to deal with changes as they appear."

Disconcertingly, nothing happened. The seasons were hot and dry; the Russian thistle barely held its own. Then man came to the aid of nature. There was a large area of recently reclaimed land, light soil and rather salty, in the district beyond Chandler. This land was largely planted to cotton. When the pink bollworm was discovered there, a non-cotton zone was established; the bollworm was evicted and a much worse pest replaced it. The Russian thistle established itself in the fallow fields and throve amazingly; it took whole quarter-sections. At the same time it had been penetrating the Valley from the north, traveling by roadsides, lodging in fence rows. Then came a favoring season and a new sort of hay-fever.

In the week that included May 1, 1931, I had 104 patients under treatment with Bermuda grass. Most of them, having begun treatment at the onset of symptoms about the first of April, were at this time quite comfortable on two or three intradermal doses a week. And then something went wrong with a third of them. Instead of the continued and increasing relief which experience had taught them to expect, thirty-three patients reported a recrudescence of their hay-fever. This did not happen all at once, but the relapses appeared a few at a time during the first two weeks of May. Also the distress of which these people complained was not constant; they might be comfortable for several days, and then have a bad day and a worse night. There was nothing in the state of the Bermuda grass, at that time declining from the peak of its pollen output, to explain their symptoms. At the same time a number of patients who had in previous years been treated for Bermuda sensitization but who so far this year had not felt the need of any treatment, sought relief from lively hay-fever symptoms.

A drive over the Valley, to see what was growing, supplied the answer. The amaranth, which seldom troubles anyone before June and which does most of its pollinating in late summer, was a month ahead of schedule. But that was not all; the Russian thistle had finally adapted itself to soil and climate and had spread widely. It was coming into bloom. All the patients who had suffered relapse were tested intradermally with amaranth and Russian thistle; all reacted positively to one or to both. So did the old Bermuda patients, now coming in for the first time this year. In addition to these, there was one patient whose only positive reaction was to amaranth, and another whose only pollen sensitization was to Russian thistle. As noted in an earlier article[2] there is a tendency to group reaction among local members of the Chenopodiales. Lamson[3], in a study of 300 patients, recently offered evidence of this bio'ogic relationship. Of the thirty-three relapsing patients, fifteen reacted alike to amaranth and Russian thistle; eleven showed the stronger response to amaranth and seven others a reaction favoring Russian thistle. The reactions were used as a guide to treatment, one or both pollens being added to the Bermuda doses as indicated by the tests. Thirty-two of the relapsing patients were relieved, in six cases by the first dose, which was the intradermal test. One patient was not promptly relieved and refused further treatment.

Fig. 5. A mass of Russian thistle or tumbleweed; the characteristics of the individual plants are not well shown.

If the agriculturists of this district knew now what they will learn later, they would by a brief concerted effort exterminate the Russian thistle. It could have been done easily any time in the past five years; it can still be done, but not so easily. Instead they will probably let the pest get beyond control and then petition for government relief. Therefore a brief description of the plant is in order. The Russian thistle (Fig. 5), Salsola kali, is not a thistle at all; it is a tumbleweed. It prefers an alkaline soil but will grow anywhere, and it may vary from the size of a blade of grass to a massive plant bigger than a wash tub. The plant has no leaves, their place being taken by spiny bracts. It is at first vividly green and succulent; as it grows, the stems become firmer. The rudimentary blossoms, consisting only of the reproductive parts, appear at the base of each spine, and new ones come out as the plant grows. The whole plant, including the pollen, contains an irritating substance allied to histamin, and the spines cause hives when they puncture the skin. The mature plant takes on a purplish color. In the fall it turns brown, breaks off its tap root and rolls with the wind, scattering seeds as it rolls. The hay-fever caused by Russian thistle is particularly severe and difficult to treat. If the plant covers the Salt River Valley, an unpleasant experience is in store for the allergic portion of our population.

CARELESS WEED AS A COVER CROP

For several years those who treat pollen disease have been puzzled by the persistence of hay-fever among a certain group of patients long after frost; in fact, it continued until the middle of December. The cause, when found, was absurdly simple.

Citrus fruit is grown in the relatively frost-free belt at the edges of the irrigated valley. Orchardists keep their groves cleanly cultivated during the summer, and they used to keep them clean the year round. Of late years, however, many of them do their

Fig. 6. Careless weed, two plants of which are growing besides a tumbleweed (Russian thistle).

last cultivating in early fall. Careless weed comes up thickly, but it grows only about a foot high. These small plants pollinate in November and in December until a killing frost. This does not interest the citrus grower, who ploughs under this cover crop of weeds to serve as humus. More and more, people of moderate means are leaving the city and building homes near the edge of the valley, or in the adjacent desert. This group furnishes the cases of late fall and early winter hay-fever, and these cases are all caused by amaranth.

MISCELLANEOUS POLLENS

A fairly frequent cause of incomplete success in the treatment of hay-fever is found in the pollen of the olive tree. In certain parts of the valley this tree has been used extensively for ornament. There are a few groves operated commercially. In April of 1931 I found it necessary to administer olive pollen as an adjuvant to other pollen therapy in eleven cases. There was one patient who had hay-fever from olive pollen alone, and this came from a solitary olive tree near her sleeping porch. All these patients were promptly relieved by the appropriate pollen therapy.

In 1922 I reported a number of cases of late summer hay-fever from the so-called "six weeks grasses", the wild grasses that lie dormant during most of the year and spring up after the summer rains. For some years thereafter there was no more trouble of that sort, for the range was over-grazed and the summers were dry. In 1931, after two wet summers, this six-weeks-grass hay-fever again appeared. The native grasses were augmented this time by the patches of Bermuda grass that are now to be seen in the hill country, even at elevations above 5000 feet. Lacking the specific pollens, I treated them with Bermuda, usually with complete success.

This outlines the changes in our local pollen picture up to date. There is no reason to suppose that it is final. Our soil is hospitable to strange plants as well as to new people and further invasions are to be expected. That is only one more reason why every doctor who treats pollen disease should educate himself to know the plants that cause it, what they are, where they grow and when they pollinate. The time and effort so spent will be well repaid by his satisfaction in treating more successfully an ailment which has become a menace to the public comfort, if not indeed to the public.

REFERENCES

1. Phillips, E. W. Ragweed hay fever in the Salt River Valley. Southwestern Medicine, 12:2, February, 1928.

2. Ibid. Hay fever in Central Arizona. Southwestern Medicine, 7:273, August, 1923.

3. Lamson, R. W. Evidence of biologic relationships among species of Chenopodiales. Proc. Soc. Exper. Biol. and Med. 28:502, 1931.

AGRANULOCYTIC ANGINA

CALVIN A. EATON, M. D.
Yuma, Arizona

(Read before the Fortieth Annual Meeting of the Arizona State Medical Association, held at Nogales, May 6-8, 1931.)

Synonyms: Agranulocytosis; malignant leukopenia.

Agranulocytic angina is an acute systemic disease, etiological factors unknown, characterized anatomically by marked leukopenia with absence of granular leukocytes, ulcerative and necrotic areas usually involving Waldeyer's ring, associated mucous membranes, and genitalia. Clinical manifestations are of hyperpyrexia, progressively lowered resistance and, usually, an early termination which is fatal.

HISTORY

The disease was first introduced by Warner Schultz in 1922. He presented five cases, describing them in detail and calling the disease agranulocytosis. The Medical World recognized this series of clinical entities as a new disease. Various case histories have been reported since the middle of the nineteenth century which we know now were typical agranulocytic anginas, because of their characteristic necrotic ulcerations of the pharyngeal membranes and other associated pathology. Since 1922, increasing numbers of cases of this disease have been described in most every country. Many of these cases do not appear to be the typical anginas as described by Schultz.

CASE REPORT

Previous History: Mrs. S., aged 38, housewife, was seen January 6, 1928, complaining of head cold with a profuse greenish purulent discharge from the nose. With argyrol nasal tampons, the condition cleared up in 7 or 8 days. Patient was not seen again until February 16, 1929, then complaining of the symptoms previously mentioned. The same treatment was instituted for about ten days and patient improved and did not return until September 16, 1929, at which time she complained of having several severe attacks of colicky pain in the upper right quadrant, with referred pain to right subscapular region. Condition was diagnosed as an acute exacerbation of a chronic cholecystitis. Her condition gradually improved under treatment but patient complained of general malaise and weakness for some time.

The blood count one month after attack showed white blood cell count of 8,400; red blood cells, 4,120,500. The patient was not seen again until June 26, 1930, at 4:30, at which time she complained of sore throat, duration two days, pain on deglutition, a duration of one day. There was general malaise, nausea, headache, small ulceration on right thigh about size of a dollar, also small ulceration on right labium majus, and pain in the right ear. Physical examination revealed slight jaundice, small ulcerated areas about one centimeter in diameter on ton-

sils, pharyngeal wall, and uvula, with marked edema of uvula. There was a small indurated area, rough and hyperemic, at site of first right lower molar, due to its extraction two months previous. An ulcerated area was found on the right thigh, approximately 3 cm. in diameter, with a red areola about 15 cm. in diameter and slight induration surrounding the ulcerated area. A similar ulcerated area, only smaller, was found on the right labium majus.

Treatment consisted of antiseptic dressings applied to the skin surfaces; painted areas in throat with one-half strength iodine. Mouth wash of Fowler's solution, glycerine, peroxide and wine of ipecac every 2 hours, alternating with a gargle containing tincture of guaiac. Rest and quiet was recommended.

On the following morning, patient did not seem to be reacting to treatment but was becoming progressively worse, so was placed in Yuma General Hospital. Major import of laboratory reports revealed the following: Urinalysis negative except for one plus albumin. Red blood count, 3,520,000; hemoglobin, 45 per cent; some poikilocytosis. White blood count, 2,850, with 94 per cent small lymphocytes, 1 per cent large lymphocytes, 1 per cent transitional, and only 3 per cent polymorphonuclears. Myeloblasts, 1 to every 100 white blood cells counted. No mononuclears, eosinophiles or basophiles. Serum reaction Wassermann, negative. Cultures: Throat and wound showed staphylococci present. No growth on blood culture.

Progressive Clinical Record: On admission, 11 a. m., June 27, 1930, temperature was 103°, pulse, 116; respirations 24. General condition became progressively worse throughout the day. The ulcerated areas in the throat had become more organized, with a profuse exudate. There was intense pain over entire abdomen, with absolutely no rigidity. Previous medication was continued and, in addition, 2000 cc. of Ringer's solution was given intravenously. On following morning, January 28, temperature, pulse, and respiration were about the same. However, tho patient complained of more pain over entire body, especially at right hip ulceration and right hypochondriac region. Patient was becoming more toxic and more critical. Additional medication consisted of 10 cc. of sterile milk given intramuscularly. In the afternoon, temperature rose to 105°; pulse, 128; respirations, 24. There was photophobia; sclera had an icteric tinge; and there was conjunctivitis, with a greenish purulent discharge. The throat area became very necrotic and sputum was a heavy mucus streaked with green. Blood count showed white blood cells, 2,500. Red blood count, same. On morning of June 29th, patient became very restless, pulse poor quality and irregular. Patient was given opiate and slept for short intervals. General condition was very grave.

White blood cell count was 2,100, a differential count showing no polymorphonuclears. Six-tenths gram of neoarsphenamine was administered.

Four doctors were called in consultation and the consensus of opinion was that the patient was in such a grave condition that only symptomatic treatment was advocated. However, 2,500 units streptococcus antitoxin were administered, and the usual 2,000 cc. of Ringer's solution and glucose was given intravenously.

June 30, lower extremity cyanotic. Patient unable to swallow and irrational at times. Pulse, weak and irregular. White blood count, 1,900; polymorphonuclears absent; all cells lymphocytes. Forty cc. of 1 per cent mercurochrome was given intravenously, together with 1000 cc. Ringer's solution. General condition and pulse were somewhat improved for a short interval. That night slight rigidity of right leg and left arm became noticeable.

Morning of July 1, white blood count was 600 cells, all lymphocytes and no polys. Subcutaneous injection of 1000 cc. normal salt solution given. Throughout the day patient was drowsy, unable to swallow; left hand twitched occasionally; pulse was irregular, breathing shallow.

In the afternoon of July 1, the white blood count had receded to 200 cells; only small lymphocytes were found. General condition remained the same until 3:45 a. m., July 2, at which time patient died.

Autopsy was not permitted.

This case, I believe, represents one of the true agranulocytosis cases as originally described by Schultz, in 1922. As I have searched through the literature of the 270 cases reported, I found about 84 with the typical text-book description, as given by Schultz. The typical cases are usually found in middle aged women; with previous history of gallbladder troubles; a sudden onset with high fever and general malaise. Ulcerations, necroses, diphtheritic or gangrenous processes—especially of the tonsils, pillars of the fauces, uvula, palate, and pharynx; and occasionally similar lesions of the gums, tongue, larynx and genitals. There is in some cases the presence of icterus. Occasional enlargement of the liver and spleen. The characteristic blood picture: A profound depression of the white blood cell count, usually less than 1000, with a disappearance of the polymorphonuclear neutrophils. There is a relative lymphocytosis. The red blood cells and blood platelets are not disturbed. The disease has a rapid fatal course.

ETIOLOGY

A great deal of work has been done to determine the etiology of the disease. In the typical cases reported, about 79 per cent have complained of gallbladder disease together with repeated attacks of tonsillitis and usually associated dental caries or apical abscesses. Several of the acute cases followed, three to five weeks, after the removal of teeth. Many believe that it is of a bacterial origin: Bacillus pyocyaneus, streptococcus, and staphylococcus are the chief organisms believed to be involved.

PATHOLOGY

The ulcerations may be deep and necrotic or superficial and cloudy, resembling a diphtheritic membrane. A marked change is noticed in the bone marrow, it being yellow and fatty, showing no cellular reaction whatever. There is a death of the granulocytes. There is cloudy swelling and parenchymatous degeneration of the liver, kidney, and spleen.

SYMPTOMATOLOGY

There is a sudden acute onset, high fever maintained throughout the disease. The patient complains of a sore throat and dysphagia which becomes progressively

worse. The ulcerative areas of the mouth and pharynx spread rapidly and may involve all of the oral and buccal mucous membrane. Some of the cases may develop jaundice. The characteristic and diagnostic findings are in the typical blood findings. A marked leukopenia is found from the first, increasing from day to day until the leukocytes are practically all absent through the course of the disease. The differential count shows a very marked decrease in the polymorphonuclears until the final stages, when they are entirely absent. There is no marked decrease in the number of red blood cells throughout the disease.

The duration of the disease is very short, usually five to eight days.

The diagnosis is fairly easily established if routine blood counts are made on cases which have ulcerative necrotic areas of the mouth and pharynx and the accompanying classical symptoms, as given previously.

The differential diagnosis should include: Vincent's angina, diphtheria, arsphenamine poisoning, radium and x-ray reactions, leukemias, anemias, septicemias, typhoid fever, tuberculosis of the throat, and other closely associated diseases.

PROGNOSIS

The prognosis is very poor. Ninety-five per cent of the cases are fatal when the white blood cell count falls below 1000.

TREATMENT

Specific treatment is unknown. X-ray and radium therapy, leukocytic extract, blood transfusion, and intravenous medication have given results where there is not a complete absence of polymorphonuclears.

CONCLUSION

This disease, which is relatively rare, should be diagnosed early, and a careful study of the etiology and treatment are most important.

BIBLIOGRAPHY

1. Hueber, Walther: No. 83824, Contribution to the problem of agranulocytoses München. med. Wchnschr., 76: 881-882, (May 24) 1929.

2. Paroulek, Jan: No. 83419, Agranulomyeloblastic septicemia cured by repeated transfusions of blood, Arch. d. mal. du coeur, 20: 648-664, 1927.

3. Zadek, I.: No. 43791. The problem of agranulocytosis, Med. Klin., 21:688-690 (May 8) 1925.

4. Lauter: No. 43790. The question of cases of septic angina accompanied by agranulocytosis. Med. Klin., 20:1324-1326, (September) 1924.

5. Elkesles, Arthur: No. 43789. Contribution to the disease picture of angina agranulocytotica, Med. Klin. 20:1614-1616 (November 16) 1924.

6. Bantz, Rudolph: No. 43788. A condition resembling leukemia, with a blood picture similar to an extreme leukopenia. Med. Klin., 19:1705-1707, (December 27) 1923.

7. Bakker, R., and Kuyer, A.: No. 101444, Agranulocytosis, Geneesk. Tijdschr. v. Nederl.-Indie, 69: 542-547, 1929.

8. Fumagalli, C. R.: No. 101516. Contribution to the knowledge of so-called agranulocytosis; Pensiero Medico, 16:149-156 (March 20) 1927.

9. Venckunas, J.: No. 101598. Agranulocytosis, Medicina 11:169-179, 1930.

10. Gaschelin, A.: No. 101599. Agranulocytic angina. Acta Oto-laryng. 12: 385-394, 1928.

11. Fieschi, A.: No. 101517. Agranulocytosis, Riforma Med. 46: 611-612 (April 21) 1930.

12. Chevallier, P.: No. 101514. Werner Schultz disease (Agranulocytosis W. Schultz, 1922). Sang. 3:317-332, 1929.

13. Reznikoff, Paul.: No. 100961, Nucleotide therapy in agranulocytosis, J. Clin. Invesitgation, 9:381-391 (December 20) 1930.

14. Felderman, Leon: No. 99206. Is agranulocytic angina a terminal stage of Vincent's angina? Eye, Ear, Nose and Throat Monthly, 9:10-11 (February) 1930.

15. Bigler, John A.; and Brennemann, Joseph: No. 97801, Sepsis with leukopenia agranulocytosis in children, Am. J. Dis. Child., 40:515-532 (September) 1930.

16. Rosenthal, Nathan: No. 97800. Observations on agranulocytosis, Laryngoscope, 40:592-597 (August) 1930.

17. Hodges, Churchill F.: No. 97627, Agranulocytosis, W. Virginia M. J., 26: 532-537 (September) 1930.

18. Gordon, William Henry: No. 92208, Agranulocytosis, Ann. Int. Med. 3:1008-1023 (April) 1930.

19. Blumer, George: No. 827207. The agranulocytic blood picture in conditions other than angina, Am. J. M. Sc., 179:11-16 (January) 1930.

20. Blanton, Wyndham, B.: No. 86757. Agranulocytosis with recovery, J. A. M. A. 92: 2099 (June 22) 1929.

21. Sharp, E. A.: No. 86731. Agranulocytic angina, U. S. Nav. M. Bull., 27:112-115, (January) 1929.

22. Feer, Waldemar: No. 46480. The question of agranulocytosis, Schweiz-med. Wchnschr., 56: 551-555 (June 5) 1926.

23. Hirdt, C.: No. 53459. Experiences in an epidemic of malignant angina, Ugesk. Laeger, 89: 186-188 (March 10) 1927.

24. Roch, M.; and Mozer, J.: No. 53549.. Angina agranulocytotica. Presse Med., 34: 1171-1172 (September 15) 1926.

25. Bautz, Rudolf: The question of agranulocytosis, München. med. Wchnschr. 72: 1200-1201, (July 17) 1925.

26. Hahn: No. 57205. Fatal monocytic angina, Deutsche med. Wchnschr., 53: 565-566 (April 1) 1927.

27. Thomson, J. J.: No. 68289. Agranulocytic angina; report of two cases, Laryngoscope, 38: 395 397 (June) 1928.

28. Haverkamp, W. C.: No. 77833. Agranulocytosis, Nederl. Tijdschr. v. Geneesk. 71: 2158, 1927.

29. Babbitt, James A.; and Fitz-hugh, Thomas, jr.: Agranulocytic angina, Arch. Otolaryng. 12:439, October, 1930.

30. Rutledge, B. H.: Hansen-Pruss, O. C.; and Thayer, W. S.: Recurrent agranulocytosis, Bull. Johns Hopkins Hosp., 46:369, Jan.-June, 1930.

31. Talley, James E.; and Griffith, George C.: Agranulocytosis, M. Clin. N. Amer., January, 1930, p. 1079.

A RATIONAL TREATMENT OF PNEUMONIA

(A Paper with a Message)

H. T. SAFFORD, Sr., M. D.
El Paso, Texas

(Read before the El Paso County Medical Society at the January 11, 1932 meeting.)

Pneumonitis, Osler's captain of the men of death, continues unabated to take its annual toll. Remedies and measures unnumbered have been advocated for the amelioration of this disease. It sometimes seems that we are about where we were when Dr. Osler sounded his caution against over-treatment, and warned the, then, younger generation of doctors that pneumonia is apt to follow an unmodified course to its termination in recovery or death, and that injudicious attempts to profoundly influence this disease, might easily occasion harm instead of benefit. In the interest of brevity I purposely discarded all reference to, or discussion of, the early history of pneumonia therapy. Outstanding remedies in the last half century that have found acceptance in the treatment of pneumonia are quinine and quinine derivatives, vaccines, phylacogens, and serums. The last named member of the latter group is the only one having a real claim to scientific consideration.

Digitalis in heroic doses has its advocates. The early and plentiful use of oxygen is increasing in favor. Osler, in his Practice of Medicine, mentions quinine as being much in vogue, but he, himself, believed it of no value as a pneumonia specific, and said there were other better antipyretics.

As to such strenuous measures as deep digitalization, profound protein shock and the like, while sometimes accomplishing seemingly miraculous cures, such remedies are to be regarded in the light of two edged swords, always fraught with the possibility of resulting diaster.

Antipneumococcic serum, in certain types of pneumonia, has proven curative beyond doubt when given in sufficient quantities. The vast majority of sufferers from pneumonia are not likely to have either typing or serum administration. Even when available, the serum treatment is by no means on a plane with diphtheria antitoxin. A more simple treatment of ready application must be selected for the average pneumonia patient. Oxygen has proven its value but its cost makes it prohibitive for the poor.

The method of treatment which I now describe is neither costly nor difficult and is within the reach of all. The hospital and the trained nurse are, of course, highly desirable, but not essential.

For years I had been using quinine in combination with other drugs in the treatment of pneumonia and, as I thought, with considerable benefit. My present method and use of this drug came about in this way. One of my patients had such persistent vomiting as to render impossible ordinary oral administration. I resolved to try giving the same medication in greatly increased dosage by enema. The results were so surprisingly good that I decided to make this a routine treatment. Just why the quinine mixture administered in this way should prove so much more curative, I am unable to explain, but repeated trials have seemed to confirm the fact that, in most cases at least, it is definitely curative.

In almost every instance, since adopting this plan of treatment about two years ago, I have found that subjective symptoms disappear, and pulse and temperature are brought to normal, or nearly normal, within forty-eight hours after beginning treatment, although physical signs in the chest may persist for a number of days, and are to be regarded as a warning that patient is not to be let out of bed too early. Cases treated include typical lobar pneumonia with extensive consolidation, tuberculous pneumonia and a number of others, probably to be classified as bronchopneumonia, in which the only findings were localized crepitant rales accompanied by cough and bloody expectoration. This list included quite a number of old people who responded as readily as did younger individuals.

I am inclined to believe that not a little of the benefit secured in these cases is the result of the consideration given the alimentary tract. The routine includes an evacuant enema every twenty-four hours and a complete denial of food the first day. Thereafter, beginning with nutriment which places the least tax upon the digestive system, a gradual increase of food is allowed as the individual shows increasing digestive capacity, the greatest care being taken not to exceed the patient's tolerance. When one considers that with the onset of a severe pneumonic process the patient is practically overwhelmed by the toxins let loose within the circulation, it is easy to conceive that the digestive processes are usually in complete abeyance. Secretions are stopped and intestinal motility is suspended or even in a condition of reverse peristalsis. If nourishment be given at this time there must result the absorption of products of imperfect food digestion which may add more than we know, to the distress of the patient.

While this treatment seems to be effective in other conditions than lobar pneumonia, I would not expect it to be of much

value in a frank bronchopneumonia, with concomitant catarrhal involvement of the rest of the lungs, nor in the influenza pneumonia, that malady which seems to respond so little to any known therapy. While this treatment, later to be given in detail, has seemed to accomplish remarkable and rather constant results up to the present, I am only too well aware that the alleged virtue of many a therapeutic fails to stand the test of more general application. One naturally prefers to wait until his position is proven beyond all doubt before making any very positive statement. In this instance, in the very midst of the pneumonia season, I am moved for several reasons to urge that a trial be given of this treatment of pneumonia.

First; because of its apparent efficacy in my hands.

Second; because of its easy application and impossibility of doing harm. Real quinine idiosyncrasy is rare.

Third; because it is a sane, rational procedure, which even if in general use, the reported curative value of the quinine enema should not be realized, the patient, because of the care given the gastro-intestinal tract, has been placed in the most favorable position to be benefitted by any further measures that may be instituted. Indeed, no symptomatic requirement should be overlooked, but should be met with common sense therapy.

For respiratory embarrassment, camphor in oil or other respiratory stimulant. Pain and excessive cough usually yield to codein in sufficient doses. Strychnia may be added if the pulse be weak. Mustard leaves and later big wet hot flaxseed poultices are also helpful in control of pain. Whiskey in reasonable doses helps to relieve cyanosis, though oxygen is superior if it can be had.

And so on, suiting each necessity as indicated.

TREATMENT IN DETAIL

It goes without saying that the patient should be given all those little aids that go to make up for him the best possible advantage. There should be a cheerful, well ventilated, but not cold room. The open window or out door plan does not lend itself to the treatment by enema. There is too much chance of chilling with the possible increase of pulmonary congestion.

A good nurse is desirable, but any intelligent attendant can carry out instructions. It should be impressed upon the patient that, though the treatment by enema is a good deal more troublesome than medication by the ordinary route, it will prove so much more effective that it is worth all the bother. And the patient should be made to understand that it is absolutely necessary that he remain as quietly as possible in bed; that any attempt to get up or even sit up, might result disastrously.

Among the little things that help the pneumonia patient is one that I believe is most important and least appreciated. I refer to the control of fear. The word pneumonia should never be mentioned in the patient's home. For the patient, the diagnosis should be made of influenza, slight pulmonary congestion or there should be recourse to some other evasion. To some responsible member of the family, the true condition should be told, but it should remain a secret between this person and the doctor. Anxious members of the family hovering about with grave faces do not contribute to the spirit of optimism which is so necessary for the patient's morale.

THE PRESCRIPTION

R.

Pulv. Ipecac et Opii gr. XII
Acetphenetidin gr. XXXVI
Quinine Sulph. gr. L
Ft. Chart. No. VI.

Sig. One powder, in half cup of luke warm water by enema every six hours.

In beginning the treatment an evacuant enema, consisting of a liberal teaspoonful of salt in a quart of warm water, is first given, to be followed one hour later by one of the powders in four or five ounces of luke warm water, administered with a large bulb syringe. It is important that the syringe be a single bulb, in fact a large sized infant syringe. If a bulb syringe be ordered often one with bulb tubing and valves may be supplied. This is entirely unsuitable, and by causing introduction of air may occasion distress to your patient and failure to get results. After the first powder, one is administered every six hours day and night, and of course, are to be retained as long as possible. The evacuant enema is to be given once each twenty-four hours and, when used, should procede medication by an hour, as in the first instance. The medicinal enemas are usually retained without much difficulty and seldom cause any discomfort. In case of any bowel irritability this may usually be allayed by the insertion of a suppository containing tannin two grains, and opium one grain. If the patient be actually vomiting, the evacuant enema may help relieve this distress by tending to overcome reverse peristalsis. Sometimes the vomiting yields to small doses of sodium bicarbonate. A teaspoonful is added to a glass of hot water and a tablespoonful of this administered every ten minutes for six doses or more.

As soon as water can be retained, it should be offered freely. Sodium bicarbonate should be then given in teaspoonful doses three times daily or one of the many proprietary alkaline preparations may be substituted if found more palatable. No other medication should be given by mouth, but all indications met by subcutaneous intramuscular or intravenous routes. If the patient has been much weakened by vomiting, the earliest nourishment should be one of the proprietary predigested food preparations. These contain partly digested proteins and carbohydrates in soluble forms, and about twenty per cent alcohol. They are readily absorbed without putting any tax upon the digestive system. As soon as may be, orange juice is ordered to be taken freely. As improvement occurs, clear broth may be given and the diet gradually increased, as tolerance is developed. Almost immediately after beginning treatment, an amelioration of distressing symptoms is experienced by the patient. After twenty-four hours, the temperature is less and the pulse rate lowered. Both temperature and pulse may be normal, or nearly normal within forty-eight hours. By this time a mild degree of cinchonism is often present, and the powders are to be discontinued for from twelve to twenty four hours, to be renewed if temperature continues. In mild cases a course of six powders is sufficient and in no case have I found it necessary to use more than the second course of six powders.

In conclusion, I would say that I think it highly improbable that this method of treatment should effect any miraculous cures in the graver types of pneumonia, but I am equally certain that it does have a definitely favorable effect upon lung inflammations and since it is certainly a safe and sane procedure, is it not worth a careful trial, even though it be a little more troublesome in application than the more usual manner of oral administration?

IMPRESSIONS OF AND SUGGESTIONS FOR STAFF MEETINGS

S. I. BLOOMHARDT, M. D.
Phoenix, Arizona

(Read by Chairman-Elect at the Staff Meeting of the Good Samaritan Hospital, on October 26, 1931).

After a period of five years of attendance at staff meetings, and after serving two years as secretary of this staff, a few thoughts or ideas for their betterment have come to my mind. The meetings at both hospitals, I believe you will all agree, have been progressively better and more interesting. The record of hospital activity which is read at the St. Joseph meeting and the review of charts of deaths given at this hospital give us a working idea of staff activity, the type of records being kept, and the degree of good, indifferent, or unfortunate work we are doing. Personally, I have derived considerable benefit from the reviews given by the chairman of the Records Committee at each meeting here and it is my hope that they will continue to be well studied, high grade, frank, and honest presentations. The case reports during the past year, with very few exceptions, have been of high standard. I think the reason for this is the fact that the doctors have been working up their cases, and writing down the principal points of interest and the discussions. Very seldom do we have the fumbling through charts, trying to find this datum or that, mixing up the history with clinical data, the physical with the operative chart, the laboratory findings with whatnot. Those who have the ability to get on their feet and review a case extemporaneously without writing it out, are very few, indeed. When one can do it acceptably, certainly it is most interesting, but for our minutes and records a written case review and discussion should accompany this presentation. During my secretaryship of two years, only on two occasions have I had any one of the staff members come to me and tell me he had an interesting case and would like to review it and have it discussed before the staff. Just the why of this I am not able to understand. I do not believe it is lack of interest, but rather due to the fact this custom has never been inaugurated and the staff members become so used to a chosen few working up all the cases and papers and being asked to do so, that when they do have an interesting case, they shrug their shoulders with a "What th' hell!" So I might suggest that, hereafter when any member of the staff has an interesting case, tell the secretary of it and in that way perhaps our case reports and papers will be even more interesting. I have tried hard to ferret out interesting cases, or cases which will bring out the most beneficial and up-to-date knowledge and discussion, but you can realize what a task this is and how often more interesting and instructive cases are passed by. It has been said that "advice is easy to give, hard to take, and usually of little value", but in regard to writing up papers, permit me to quote from one of my former surgery chiefs and associates, Dr. Walter Estill Lee: "I would advise the younger men to write papers, and in writing bear in mind what the old minister said, 'Few souls are saved after the first fifteen minutes of the sermon'. Write the paper not to show

how learned you are, or to show the high type of man who may be in your audience that you are in his class, but rather try to tell those in your audience, who may not know as much about the subject as you do, something that may interest and help them. Do not try to make too many points. The late Dr. A. J. Ochsner used to say, 'If I can carry two lessons in each paper, it is about all the average man, whom I want to reach, can absorb at one sitting; anything over five would be lost.'

"In urging you to write, I do not wish to stress the idea that your papers will be valuable to anyone but yourself, but they will be of incalculable worth to you because they will crystallize your ideas and will bring forward questions you have not answered and must study further.

"In delivering papers and addresses, begin by presenting them before small societies and work up to the national organizations; you will find men in small societies equally as bright and eager to learn as you will find in the large societies."

You will all agree this is sound advice and none of you have ever prepared a case report or paper without it being of incalculable worth to you and of some worth to the average of us. So, again, I would like to urge any or all of you to let your staff officers know of anything of interest and, if it is their desire, they can put in on the program.

The increasing frequency of lantern slides to demonstrate findings or show us figures along with the papers also has a tendency to make our meetings more scientific and valuable. We decided during the past year to make our meetings more open and our constructive criticism more direct, but this has never been carried out, probably because of fear of causing enmity between staff members and of interfering with the financial stability of the hospital. Personally, I wish we could work out a friendly method of checking up and helping those of us who are not doing the best work and cheering those of us who are doing very good work, for their efforts. Progress is the watch-word of science and it is undoubtedly an important quality of mind in seeing one's period and one's work in its proper perspective. I think if it were possible to have every member of the staff, for example, as chairman of the Records Committee for one month, it would be a good thing. When you review the case records of the month and find one man has a high mortality in some one pathological condition and go back over the card index of records for months back and find he has been having that same bad record, should there not be a way we can help him and, if we cannot help him, do something to help society as a whole? When you study the card index and find the mortality for this operation and that operation much above the average mortality of the medical centers, it seems to me again, we should all try and help. This state of affairs is not only a local one. It was my privilege to have dinner with Dr. Bloodgood the past winter during his short stay in Phoenix and after dinner, in our conversation, he abruptly asked me two questions: first, how honest was the surgery and medicine of the Southwest; and second, would I consider the surgery good surgery? And he told me that all over this country this was one of the big questions of the profession and among the educated lay people. Willis and Beaver tell us that surgeons can no longer ignore the fact that the surgical judgment, in particular, and the surgical technic outside the great clinics is "almost criminally defective". In an area where the vital statistics had been kept accurately it was found that the deaths from appendicitis per 100,000 population have increased 31 per cent between the years 1905 and 1922 and during the same period there has been an increase of 77 per cent in deaths from gallbladder disease and 250 per cent in deaths from goitre. It is unfair to credit all of the increasing mortality to poor surgery, but we must admit that, in the later years, these conditions are being more generally and more radically treated by surgeons than in 1905, and it is certainly probable that the increased mortality is, to a large measure, due to injudicious and unskilled surgery. Such statistics should awaken in the conscience of every one of us a new sense of responsibility. Notwithstanding the advance in knowledge of anesthesia, our increased knowledge of function and pathological changes, our ability to detect pre-operatively surgical handicaps, and our increasing knowledge of post-operative complications, statistics show this enormous increase in the mortality rate of surgical diseases. But, unfortunately the mortality rate in surgery is not the only indictment which we must bear; I refer to those cases all of us examine after they have been operated upon once, twice or more times, and find the results of surgical intervention most disappointing. There is most always in these cases, evidence to show that the reason operation fails to afford the patient symptomatic relief is that the fundamental cause of the trouble has not been discovered and removed. The feeling is rapidly growing

that isolated lesions in any one organ rarely occur. This group of unimproved and disabled operative cases was called by the late Dr. Joseph Price "surgical junk". Although the exact relationship between disease changes in one section of the gastro-intestinal system with those in another is not known, the feeling is growing that isolated lesions in any one organ rarely occur. Biliary disease, for instance, is invariably associated with disease of other viscera, especially the liver, kidney and pancreas. And so with other systems of the body; there is a relationship between them and they are gradually being worked out and demonstrated by our scientific leaders.

The excuse for increasing mortality and disability cannot be for lack of material equipment because in our country, even in the smaller communities, the hospital equipment and the surgical opportunities are beyond criticism. Monihan tells us in one of his lectures that surgery is not learned easily, the training is hard and lasts a man's lifetime. "It must be under a master's eye and be influenced by his criticism not less than by his spiritual encouragement. It must not be light heartedly or recklessly undertaken nor can it be a matter of display. Since the war, especially, the incompetent and ill-trained surgeon is allowed too free a hand and enjoys too wide a scope. The methods of surgery learned in the war do not fulfill the needs of civil life. The emergencies of war, the hates of war, and the sudden and heavy pressure of urgent work, made it necessary to have operators at once available. There is today too much bad surgery; surgery performed by those who have, perhaps, some natural skill but which has never been trained and moulded to right practice. In surgical work, craftmanship is much and knowledge is much, and wisdom, which is the timely and rightful application of knowledge, is more, but as we establish our place in the world, it is chiefly character that counts."

But I have digressed to a considerable extent from the purpose of this paper. I gave notice it was to be a "hot weather paper"; by that I meant a rambling, easy-to-write affair, in which I could jump about from point to point without particular reason or thought, nor has anything I have written been written in the sense of criticism either local or general, for the big reason I fear my own work would not warrant criticism of that of others. But in concluding I hope our staff meetings become somewhat less formal, that discussions or criticism be entirely constructive and lose the sting they occasionally possess, that advice be more frequently given or ideas suggested by those who have had broader and more recent training and bigger experiences, and this advice or suggested ideas be given in a friendly manner and be accepted as such. In a small community, such as this, it is very hard even with considerable reading and visiting of clinics, to keep abreast with the best in medicine and surgery, but I do think we can help each other more right here in the staff meetings, and by doing that, help society at large.

A CASE OF DISSECTING ANEURYSM OF THE AORTA

DAVID M. DAVIS, M. D.
L. B. BALDWIN, M. D.
PAUL GUTTMAN, M. D.
Phoenix, Arizona

(Presented to the Staff Meeting of the Good Samaritan Hospital, Phoenix, Ariz., Oct, 1931.)

L. H. H., male, aged 55, entered the hospital at midnight of Sept. 17, and died the next afternoon at 3 p. m.

His health had always been very good without serious illness. About two weeks before entering the hospital, he began having headaches daily. Three days previous to his admission, he went on a squirrel hunt, and during this expedition, while hunting for game, he was suddenly seized by an acute attack of pain between the shoulders and upper back. A passerby found him in a state of collapse and summoned the family to whom he complained of this intense pain, of weakness and of a general feeling of numbness. He was nauseated and vomited persistently. A local doctor examined the urine and told him that he had Bright's disease. The next day, his condition remained about the same, though on the third day, he began feeling a little better for a short time. Ever since the onset of his illness, he had passed very little urine. There had been no vertigo, tinnitus, diarrhea or indigestion. The past history was essentially negative except for the fact that he had had night voidings for several years. There was nothing of significance in the family history.

Physicial examination showed a well nourished, muscular, middle aged man, who appeared drowsy and stuporous, but could be aroused and answered questions intelligently. He complained of almost total blindness. The temperature was 99, pulse 112, respirations 30, blood pressure 244/60. The skin was rather deeply cyanosed and there was slight clubbing of the fingers and toes. The pupils were constricted but reacted to light. Rough neurological examination was entirely negative. The teeth were in wretched condition, many missing, some carious stumps. There was retraction of the gums and marked pyorrhea. Patient breathed normally except for slight increase in rate and depth. The lungs were clear. The heart was enlarged to the left, action forcible and regular, and there was a ringing, accentuated second aortic at the apex. There was a musical, mid systolic murmur, transmitted into the left axilla. The peripheral arteries were all markedly thickened. The abdomen, genitals and extremities were all normal.

Laboratory studies showed a leukocytosis of 19,500 with 87 per cent polymorphonuclears, a urine of high specific gravity, loaded with albumen, casts of all description, white and red cells. The NPN was 75 mgm.

The diagnoses made at this time were: acute nephritis, chronic arteriosclerotic nephritis, apical

arteriosclerosis, chronic myocarditis, uremia, hemorrhage in unknown locality.

On the night of admission subcutaneous infusion, hot packs, and cathartics were ordered. He was catheterized but no urine obtained. The next morning however, following the continued forcing of fluids by mouth, by colon irrigation, and by infusion, he voided liberal amounts of smoky, rather highly colored urine.

The patient was again seen the same day. He gradually became more stuporous, his pulse became rapid and thready, the blood pressure dropped progressively, and he finally lapsed into coma and died apparently of circulatory failure.

Autopsy: (Dr. David M. Davis). The exterior of the body showed nothing of special note. There were no wounds or injuries, no edema and no disproportion between the upper and lower parts of the body. The muscles were very well developed, the man evidently strong and robust. The abdominal contents appeared normal, except that the appendix was completely retrocecal. It showed no signs of inflammation. The bladder was moderately distended, containing about 500 cc. of urine. On removing the sternum, it was noted that some of the mediastinal fat was suffused with blood and further examination showed that the upper mediastinal tissues were thickened and ecchymotic. This ecchymosis was most pronounced on the left side and continuing down past the hilum of the lung, as far as the diaphragm. The heart was markedly enlarged and firm. The pericardial sac was smooth and normal in appearance, but containing a moderate excess of blood tinged fluid and near the point where the pulmonary artery disappeared, a little of the ecchymosis could be seen on its interior surface.

Both lungs showed very marked anthracosis, but were not adherent anywhere and the pleurae were smooth with no excess of fluid. Inspection, however showed a number of small petechiae on the parietal pleura on both sides. Both lungs, on removal, were everywhere crepitant with no nodules or areas of induration, and no evidence of edema. The heart was about twice normal in size, the increase being due to a tremendous hypertrophy of the left ventricle. The tricuspid and pulmonary valves were normal. The mitral valve showed a definite, but moderate nodular thickening along its free edge. The aorta was slightly thicker than normal and showed a moderate degree of atherosclerosis with superficial yellow plaques. This atheroma involved the aortic valves to a slight extent, but apparently not enough to interfere with their competence. The coronary arteries were thickened, but everywhere patent. The myocardium showed a few small gray fibrotic areas; they were not adherent plots. The small and large intestines were normal. The spleen showed a slight degree of acute congestion with a grayish red pulp, which scraped off easily. The liver was of normal size and appeared with no evidence in cut section of fatty degeneration or necrosis. The gall bladder was normal in appearance with no thickening and no adhesions. It contained green bile of normal consistence, which was pressed easily into the duodenum. The pancreas was normal. The stomach and duodenum were normal.

Attention was then turned to the mediastinum. The pulmonary arteries were normal. The main bronchi and trachea were normal. The aorta was then slit up and it soon appeared that the ecchymosis was greatest about the aorta. In removing a segment of the descending arch for microscopic examination, the scissors passed into a good sized cavity filled with blood clot and it became apparent that there was a dissecting aneurysm of the aorta. Unfortunately the amount of cutting which had been done on the aorta made it impossible to discover with certainty, the point of rupture. Careful probing then showed, that the cavity surrounding the aorta, extended up to, including part of the arch and a small portion of the innominate artery. Both common carotids were normal, downward the cavity was probed as far as the root of the diaphragm, and where the aorta lay free (entirely) in the cavity. The entire mass of prevertebral tissue including the kidneys was then removed from a point just below the bifurcation of the aorta to a point just below the larynx. Dissection of the renal arteries showed that they were normal up to the point of origin in the aorta on both sides, but the abdominal aorta at point about 5 cm. below renal arteries was surrounded by a cavity containing a thin layer of blood clot. Further dissection was postponed until after fixation. The kidneys were removed from the specimen, the fatty capsule on each side was normal. The true capsule was slightly adherent throughout. The surfaces of both kidneys were slightly irregular, paler than normal and covered with a multitude of fine petechiae. They were about normal size, the left somewhat larger than the right. On cross section, the pelvis was normal, except for excess of peripelvic fat. The cortex was markedly thinned averaged about 4-5 mm. in thickness, and showed a fine mottling throughout. The general effect was about normal color, but close inspection showed that there was a pale background with many tiny areas of hyperemia. The ureters were normal on both sides. The bladder was thin, the surface smooth and pale. The trigone was Y shaped and not elevated. The ureteral ordifices were normal in size, position and appearance. The vesical orifice was normal in appearance, but showed moderate contracture, not admitting the tip of the little finger. The prostate and rectum were removed in one piece. The rectum was normal, the prostate appeared normal externally, but dissection was postponed until after fixation.

Anatomical diagnosis. Arterio-sclerosis with hypertension. **Rupture of the thoracic aorta. Dissecting aneurysm of the thoracic and abdominal aorta.** Hypertrophy of the left ventricle, chronic mitral endocarditis, chronic myocarditis (slight), chronic nephritis (arteriosclerotic), acute ischemic nephrosis, contracture of the vesical orifice (slight), retrocecal appendix.

Microscopic examination, by Dr. Paul Guttman (only sections of the kidney and aorta are here considered).

Kidney: There is a marked congestion of all capillaries and veins throughout the sections examined. The capillaries of the glomeruli are markedly distended. The proximal and distal convoluted tubules show a marked degree of hyaline granular degeneration. The small arteries show marked thickening of walls. There is a moderate degree of subendothelial deposit of hyaline in the small arterioles. There is no evidence of recent or old inflammatory changes.

Aorta: Sections through various portions of the abdominal and thoracic aorta show the following changes: There is a splitting of the media at about the junction of the inner two-thirds and outer one-third. The space between these portions is of varying size and contains blood clot. A thin layer of recently formed thrombus is present over the exposed surface of the media. Immediately beneath this thin layer of thrombus is a moderate reaction of polymorphonuclear and an occasional lymphocyte. The vessels of the adventitia are markedly hyperemic and there is also a moderately acute vascular

reaction in this layer. There is no evidence of perivascular infiltration of lymphocytes in the vasorum. The media appears normal, other than the changes described above. The intima in most places is markedly thickened. There is a marked increase of hyaline connective tissue which, in places, shows a marked degree of fatty degeneration.

Impression: The examination of the vessel walls indicates no evidence of syphilitic involvement. There is a marked degree of atheromatous degeneration of the vessel which might be considered in the etiology of the dissecting aneurysm. The question of a mesarteritis must be considered, as this condition is often thought to precede the formation of dissecting aneurysm. The marked congestion and early degeneration in the changes of the kidney could be well explained on the basis of vascular disturbance caused by spreading of the dissecting aneurysm about the vessels of the kidney.

DISCUSSION

DR. DAVIS: These findings explain perfectly all the symptoms of the final illness. The sudden attack with pain between the shoulders, numbness, weakness, faintness, nausea and vomiting. In retrospect it appears to me that there may have been some disproportion between the circulation of the upper and lower extremities. The radial pulse was full and bounding, while the feet, as I remember, were slightly pale and a trifle cyanotic. Had one had this extremely rare condition in mind, it might have been possible to guess the correct diagnosis. The case, however, is a beautiful example of the importance of doing autopsies, and is also another example of the various lesions which may give a picture simulating acute nephritis.

DR. BALDWIN: There are certain features in this case that are of great clinical importance because they point definitely to a syndrome, which although extremely rare, is as striking and characteristic as the clinical picture of coronary occlusion. Now that all evidence can be viewed, it is clear that the true diagnosis should have been suspected from the beginning. I have only seen two cases of dissecting aneurysm that came to autopsy, and in each of them, the wrong diagnosis was made during life. In one coronary occlusion was suspected, in the other acute pancreatitis.

In this case there were indications strongly suggesting a vascular accident. (1) In the first place, the patient had been perfectly well and suddenly developed an acute pain in the back and went into collapse. This in itself is certainly not suggestive of acute nephritis but rather of a hemorrhage or coronary disease. (2) In the second place, the pain was between the shoulders and in the course of the thoracic aorta. (3) In the third place, there was fever and leukocytosis, which almost always accompanies any vascular accident. (4) And in the fourth place, there was widespread evidence of vascular damage such as thickening of the peripheral arteries and hypertension. With these points properly correlated, the urinary evidence of acute nephritis should have actually assisted in making a correct diagnosis, instead of throwing one off the track, because with the other points in mind, it should have suggested a hemorrhage in which the kidneys were affected. This condition could only be explained by pressure on the renal vessels so as to produce an acute ischemia. This is of course, exactly what occurred. The dissecting aneurysm by extending beyond the renal arteries caused pressure to be exerted upon the vessels, thus interfering with the blood supply to the kidneys.

I have seen one condition in which a similar picture of nephritis was produced by pressure on the renal vessels, and in which the diagnosis was only made at autopsy. In this patient however, the symptoms were gradual in onset and the nephritis was of a more chronic nature. The patient in a matter of months went through all the stages of failing renal function, and finally died of uremia. Autopsy revealed Hodgkin's disease involving the retroperitoneal nodes. These enlarged nodes produced marked pressure on the renal vessels so that there was an inadequate blood supply to the kidneys, resulting in renal insufficiency.

In conclusion, it may be emphasized that when a patient has hypertension and general arteriosclerosis, any sudden train of symptoms, followed by circulatory collapse should strongly suggest hemorrhage or coronary occlusion. If such an individual also has evidence of acute nephritis, then a dissecting aneurysm of the aorta should be suspected.

It is important to differentiate clinically dissecting aneurysm of the aorta from acute nephritis, because the prognosis is very different in the two conditions, the former being hopeless and the latter having a fair chance of recovery.

RADIOSENSITIVE TUMORS

HARLAN P. MILLS, M. D.
J. M. GREER, M. D.
W. WARNER WATKINS, M. D.
Phoenix, Arizona

(Presented and discussed at the Staff Meeting of St. Joseph's Hospital, Phoenix, Ariz., June 8, 1931.)

Case 22637: School girl, age 7, entered hospital May 14. History of good health until May first, when she had some pain in abdomen lasting a few hours; two days before entering hospital she had a recurrence of the pain. Dr. Penn of Lichton was called. He found an abdominal tumor and referred child to Dr. Greer who placed her in the hospital for study. Examination showed an elongated mass in the right side extending downward toward pelis; it was not tender, was somewhat movable, and was apparently notched in the middle. Girl was in no pain nor shock. X-ray examination for possible bowel obstruction was made without revealing evidence of this. During this examination heavy lung densities were found which had the characteristics of metastases. Urosel ectan showed marked dilatation of right kidney pelvis and calyces and displacement upward of kidney. Urine and blood were normal, except for 10 to 15 pus cells per high dry field. Temperature ranged from 99 to 100, pulse 100 to 120, respirations 25 to 35.

Conference of Cancer Committee was called and they concluded that surgery was not advisable, and that radiation should be tried.

DR. GREER: Here is a child eight years old presenting a large indefinite mass in the abdomen. What is it? The history does not help a great deal. She had a little pain two weeks previous to the time first seen and was slightly ill, but not enough for a physician to be called. When seen she complained of pain in the right lower abdomen, severe enough to cause her to cry. No further history could be obtained and no past history of importance.

Upon physical examination the child did not look sick. She was somewhat undernourished, the heart sounds were normal, there were some indefinite rales in the chest. Upon inspecting the abdomen the musculature stood out prominently but she lay with the thighs and legs extended. Upon palpation one was first impressed with the hardness of the abdomen and upon deeper palpation a large hard mass could be made out, extending from the liver area

down the right side into the groin and over to the left side below the umbilicus. The mass was slightly irregular, not nodular, and presented a definite notch, or what felt like a notch. The lower part gave a rather cystic feel although very tense. Bimanual palpation with one hand over the loin and the other over the abdomen gave the sensation of a large continuous mass slightly movable. It could be felt distinctly from behind between the spine and the twelfth rib.

The various tumors of the abdomen that come to mind in the differential diagnosis of this case are:

1. Polycystic kidney.
2. Hydronephrotic kidney.
3. Hypernephroma.
4. Sarcoma of the kidney and retroperitoneal sarcoma.
5. Tumors of the gall bladder and liver.
6. Tumors of the cecum and colon.
7. Cysts and sarcoma of the ovary.

Upon further study it was found that there were peculiar dense areas throughout the lungs, shown by the x-ray, that were interpreted as metastases from some malignant tumor. This immediately ruled out, in our minds, all the benign tumors mentioned above, such as ovarian cysts, polycystic and hydronephrotic kidneys, etc. This then left us with the malignant tumors of the abdomen. Where did this tumor arise? Geographically, this tumor was in the region of the right kidney. Upon examination with uroselectan, some disturbance in the normal appearance of the kidney and particularly the pelvis was found. This leaves us with the most likely diagnosis of a malignant tumor of the right kidney. However, we must not be too sure of this, as it is possible to get a disturbed kidney pelvis and circulation, from tumors outside the kidney.

What are the malignant tumors of the kidney? Hypernephroma, sarcoma and a myxo-sarcoma of the fatty capsule of the kidney. Several years ago Kerley of New York had the following to say about malignant tumors of the kidney in children:

"Adenosarcomata and adenocarcinomata have been described and the degree of malignancy cannot be determined by their histologic structure. Malignant neoplasms of the kidney are more common before the fifth year of life than in any succeeding decade. The tumors have been classified as carcinomata and sarcomata. Most of the growths, however, are atypical mixed tumors of embryonic origin, and may contain striped muscle, cartilage, and lipomatous or fibrous connective tissue. To such forms the term rhabdomyosarcoma has been applied. The hypernephroma is derived from suprarenal tissue, which may be in the developing kidney. This tumor is subject to great variations in size and structure, and may resemble sarcoma, adenoma, carcinoma, or perithelioma. The growth characteristically contains pigment, which is identical with that found in the adrenal. Not infrequently the hypernephroma becomes cystic. Malignant growths of the kidney often attain enormous size, half filling the abdominal cavity and displacing certain of the contained organs. The abnormal mass is usually movable and occasionally communicates pulsations from the subjacent aorta. The edges of the tumor are more rounded than those of an enlarged spleen or liver, and the anterior surface is less closely related to the ribs. Apart from the local physical signs, the patient may present no significant symptoms. Nutrition, however, is generally impaired, and in many instances the tumor occasions dragging pains and hematuria."

Coming back to the differential diagnosis of this case.

You may ask why a pyelogram was not done. Drs. Ketcherside and Shields did not feel that enough additional data and information would be obtained to warrant the procedure. If there had been hematuria we could have been sure that the tumor involved the kidney, but so far as we could determine there was no hematuria. In a child it is sometimes impossible to distinguish a renal swelling from a sarcoma of the retroperitoneal cellular tissue, situated on the median side of the fatty capsule of the kidney. Usually these tumors lie nearer the median line and cause but little displacement of the colon. However, when they are very large it is quite difficult to differentiate them.

Sarcoma of the ovary can usually be traced to the pelvis. In this case it seemed that the tumor was more associated to the region of the right kidney than the pelvis.

In this case it was difficult or impossible to palpate or percuss a line of demarcation between the liver and tumor. There was more bulging in the lateral abdominal wall and also posteriorly than would be expected in a tumor of the liver.

In so far as the diagnosis before any treatment was instigated it would seem to me to rest between adenosarcoma of the kidney and hypernephroma.

Case 22754: Young Mexican man, age 19, student. First seen by Drs. Carlson and Gaede of Jerome about April 10th, at which time he complained of shortness of breath and cough of about a week's duration. He was unable to sleep on his back on account of dyspnea and some pain on coughing. The dyspnea grew steadily worse and cough more aggravating, and neck began to enlarge. At time of first examination he had lost 12 pounds in weight and had lost strength perceptibly. Lymph nodes were palpable in neck and groin, with chest dullness anteriorly to mid-clavicular line. X-ray on May 14 (at Jerome) showed a large tumor in posterior mediastinum, extending to mid-clavicular line on each side and not visibly pulsating when observed under flucroscope. Lymph node excised and sent for pathological examination. Urine negative. Blood: On May 16, reds 5,170,000, whites 28,700, about 50 per cent polys. Hbg. 90. On May 18, whites 33,600. On May 19, reds 4,800,000, whites 54,500, 20 per cent polys and about 70 per cent premature lymphocytes, platelets scant. Patient was sent to Phoenix for advice as to radiation treatment and entered this hospital May 20. Blood count here was 44,300, 80 per cent lymphocytes, 18 per cent polys, 2 per cent eosinophiles.

DR. MILLS: The summary given contains practically all the positive findings in this case, both from the laboratory and physical examinations, except for report on tissue specimen which was a cervical lymph node, removed while in Jerome and reported upon before the patient came to Phoenix, as follows: "Sections show densely packed small round cells with deeply staining nuclei and without definite arrangement. We would consider as a new growth, evidently some type of lymphoma; the question of malignancy not definitely determined. Are there any abnormal blood findings?"

The diagnostic problem apparently limits itself to the question of the nature of the tumor formation in the mediastinum and associated enlargement of lymph nodes in neck and groin. Tumors of the thymus occur which give an outline on an anterio-posterior roentgenogram identical with that shown on the first film of this case. Thymomas of lymphosarcoma type are reported which metastasize to the lymph nodes, both the cervical and axillary. The tumor in this case is in the posterior mediastinum which would eliminate the question of a thymoma.

The large size of the mediastinal tumor and the relatively slight enlargement of the cervical and inguinal lymph nodes suggest that the primary

growth is mediastinal. This is supported also by the presenting symptoms of cough and dyspnea.

Considerable difficulty is often encountered in the diagnosis of lymphoid enlargements, for the reason that this tissue responds to many influences, such as irritation from toxic substances, show hyperplasia from inflammation, and in the leukemias, as well as the hyperplastic changes due to involvement in new growth. Also the cells of lymphoid tissue are quite mobile as compared with most of the fixed tissue cells of the body. So that tumor processes tend to spread diffusely and quite promptly to other lymph nodes.

The most common cause of lymphoid enlargement is tuberculosis which induces overgrowth of all the cellular elements, the lymphocytes, reticulum cells and endothelium. It is sometimes difficult to determine when we are dealing with a granuloma or a new growth but in this case the evidence at hand would seem to exclude tuberculosis or other type of granuloma.

Simple lymphoma or hyperplastic lymphadenitis is usually local or confined to one region of the body, is more or less self limited and at no time leads to systemic invasion. After a period of growth these enlargements often remain stationary over a period of months or years and show little or no tendency to necrosis. Their cellular structure shows an increase in size and number of lymph follicles and overgrowth of small lymphocytes and there is absence of rapid growth, infiltration beyond the capsule, mitotic figures, atypical cell forms, and other signs of malignancy which differentiate from lymphosarcoma.

Lymphoid hyperplasia also occurs in the various phases of leukemia. Here we find a generalized involvement of lymphoid structures of the entire body as well as systemic involvement of the blood forming organs. There is a tendency for lymph node chains in various parts of the body to become enlarged at the same time and with practically simultaneous enlargement of all the nodes of a chain or group. In leukemia we expect to find marked and characteristic blood findings, so that a diagnosis is usually evident from the blood picture alone. In this case we find blood changes which are somewhat confusing. The total leukocyte count up to 54,000 and the differential showing a marked preponderance of lymphocytes with premature cell forms. However, in the absence of knowledge of extent and distribution of lymph node enlargement and the result of biopsy we could hardly be justified in making a diagnosis of leukemia. It is possible that as further development of the lesions and a continued study might change our conclusions and we believe that lymphatic leukemia in an early stage of its development might well be retained as a diagnostic possibility.

Hodgkin's disease, lymphogranuloma or lymphoma malignum, as it is variously named, must be given serious consideration. It is not at all unusual that the lymph node enlargement in Hodgkin's disease be confined at first to a definite group, such as the mediastinal and gradually spread to other parts of the body and often respiratory difficulty brings the patient to his physician. In such cases a bulky mediastinal tumor is formed, causing marked pressure symptoms. The blood picture is not diagnostic but in well developed lesions the microscopic tissue section is quite characteristic. There is found an overgrowth of reticulum cells, together with large and small lymphocytes, plasma and endothelial cells and eosinophile cells. The proportion of these cells varies, due probably to the stage of development of the process. Frequently the cell structure resembles lymphosarcoma and recently Levin has published a paper suggesting that these two conditions are practically identical. After a very complete study of more than 500 cases he has "gained the conviction that lymphoma malignum (Hodgkin's disease) and lymphosarcoma are nearly identical in their clinical manifestations and that they both represent a special type of malignancy." Clinically the end result is similar as a fatal termination usually ensues, after remissions and exacerbations over a varying period of time. Other writers, among them Kundrat, has noted the development of lymphosarcoma from growths considered as lymphoma malignum.

In the case presented we are suggesting a diagnosis of lymphosarcoma. The evidence is possibly incomplete in some respects and perhaps after observation for a period of weeks or months and further development occurs, our conclusions may have to be changed. Mentioning a few points which suggest this diagnosis we find that we are dealing with apparently a new growth confined to lymphoid structures; that it has arisen from a group of lymph nodes, rather than from single discrete nodes over the body; that it occurs in a robust male in early adult life which is a most common period of development of this tumor, and also that the growth is apparently spreading along lymphatic channels, rather than by local infiltration.

The single biopsy specimen, while not conclusive, shows cellular structure consistent with this diagnosis. There is found a diffuse hyperplasia of lymphoid cells, mostly small, but in part large round cells, many showing hyperchromatic nuclei and along the capsule of the node there are pockets of atypical cells which have broken through and occupy irregular spaces beyond the lymph node capsule. The blood picture is consistent, if our interpretation is correct, in that the excess of leukocytes and presence of immature cells represents an escape into the blood stream of atypical and hyperplastic cells from the newgrowth by means of the lymph spaces adjacent to the involved lymphoid structures. And, lastly we wish to suggest the therapeutic test; that of the effect of radiation upon this tumor.

If, after proper application of radium or x-ray there is not a prompt and very marked reduction in size of this new growth we would be inclined to change our diagnosis in the face of histologic, clinical and other evidence presented, since it is known that lymphosarcoma is radiosensitive to a greater degree than any other tissue.

DISCUSSION

DR. WATKINS: These two cases of tumor growth are interesting and instructive from the standpoint of radiosensitivity of tumors, so that it seems worth while to present a few of the well known facts about sensitivity to radiation. The whole practice of radiation is founded on the actual and relative radiosensitivity of tissue cells, so that the first and most fundamental consideration in any condition to be treated by radiation is the estimation of the relative sensitivity of the normal and pathological cells in the area under consideration. It was only after many disastrous experiences with radiation that workers in this field established the comparative sensitiveness of normal tissue cells, and because the skin was the organ which suffered most in these experiences, the epithelial cells of the skin are taken as the basis of comparison for radiation dosage. If we take the sensitivity of the skin as the unit (1), the relative sensitiveness of the other body cells can be graded in accordance with the fol-

(Continued on page 89)

PUBLIC HEALTH NOTES

J. ROSSLYN EARP, DR. P. H.
Director New Mexico State Bureau of Public Health

SICK SCHOOL CHILDREN

February is the month of all months when children are kept out of school by sickness[1]. During the seventeenth fiscal year, attendance in the schools of New Mexico was 69.5 per cent enrolled[2]. Of all the United States, Oklahoma alone had a worse record. The cost of the schools to the taxpayers of New Mexico during this year was $5,133,324. The taxpayers thus paid $1,565,664 for teaching children that were not there. It is not known what proportion of the New Mexico absentees were kept away from school by sickness. The study made by the U. S. Public Health Service in Hagerstown, Maryland[3], showed that 57 per cent of absenteeism was due to sickness and 43 per cent to "other causes". If we apply these proportions to New Mexico, we find that sickness of school children cost the New Mexico taxpayers more than $800,000 in a single year, or more than $2.00 per capita.

A discussion of these findings was released to the press of New Mexico on December 17 with the following comment;

"It is a striking fact that less than one fifth of these illnesses are seen by a doctor. How much time might be saved if a doctor were always consulted for small complaints—and lives too! The 'sore throat' that turns out to be diphtheria could be treated in time. Small cuts would not become infected. Fewer colds would become bronchitis."

If, as a result of this publicity, some doctor is called away from a waiting meal to see a school child with stomach ache we hope that we may be forgiven. To an experienced physician a stomach ache is often quite plainly just a stomach ache and the more or less innocent cause may be just as plain. It is important always to bear in mind that to the parent an acute appendicitis may equally seem to be just a stomach ache, and to the parent it is worth while to have many unimportant ailments properly labelled by the physician—and to pay adequately for the labels—in order that, once in a while, important mischief may be scotched at the outset.

It is interesting that in Hagerstown only 18 per cent of the childrens' illnesses received medical attention. Of course, some complaints received medical care with greater frequency. Thus, a doctor was called to every case of pneumonia and of diphtheria. But the statistics here may easily be giving a too optimistic conclusion, for the diagnosis of pneumonia or diphtheria would not often be made without medical assistance. Only one fifth of the sore throats were seen by a doctor, an who is to tell us that there were no cases of diphtheria or scarlet fever among the other four fifths?

WRAPPED MILK

An interesting visit of the English Society of Medical Officers of Health to the Bladen Dairies at Blandford in Dorsetshire is reported in a recent Lancet[4]. Here some 20,000 gallons of milk are collected daily from ninety farms. As the milk comes in, it is tested and graded. The twelve farms which score highest each month get one cent per gallon extra for their milk, and the next twelve get a half cent extra. The milk is pasteurized with precaution by the holding method, before it goes out to London and Bournemouth to be sold. But the most interesting item is the wrapping in which it is dispatched, namely, in paper cartons. The weights of a half-pint bottle and of an equivalent carton are respectively about 12¼ ounces and ¾ ounce. The approximate cost of a carton is one cent and "breakages" amount to only ¼ per cent. Small cartons containing one third of a pint and intended for school children are retailed at two cents. The chief interest of this scheme to health officers lies, of course, in the fact that the container is sterile when loaded and that it is never used again.

Some American readers may be surprised to learn that Sir Ernest Debenham, who owns these dairies, got his carton idea from the United States.

SPOTTED FEVER VACCINE

The Hamilton Laboratory at Hamilton, Mont., is able to supply vaccine for immunization against Rocky Mountain spotted fever to physicians living in infected territory. Dr. R. R. Parker requests that the following points be observed in regard to its use:

1. Requests for vaccine should be addressed to the Officer in Charge, United States Public Health Service, Hamilton, Montana.

2. Each request for vaccine should specify either the number of persons to be vaccinated or the number of cubic centimeters needed on the basis of 4 cc. to the person.

3. Vaccine is furnished to physicians without charge and the fee for administration should be nominal.

4. The vaccine is expensive to manufacture and amounts requested should be limited to use which can be foreseen.

5. Reports received at the Hamilton station suggest that, if vaccine is administered soon after a bite by an infected tick, a considerable amelioration of symptoms and shortening of the course of infection may result. Physicians will therefore be justi-

fied in recommending the taking of the vaccine by tick-bitten persons as soon as possible after the bite is received.

6. Therapeutic use of the vaccine after onset of symptoms is not recommended. In sections where the less fatal types of infection occur, some physicians have so used it with supposedly good results, but the difficulties which prevent certainty on this point can be readily understood. Results in the highly fatal Bitter Root Valley cases have suggested that, in the more severe type of the disease, its use may even be harmful.

7. It is especially requested that the Officer in Charge of the Hamilton laboratory be advised of any cases of spotted fever which may occur in vaccinated persons and that as detailed a clinical record as possible be kept of such cases.

1. Collins, S. W. The Health of the School Child. Public Health Bulletin No. 200. Washington, 1931. Pp. 159. Price 35 cents.
2. Statistics of School Systems, Bureau of Education, 1930.
3. The Lancet, 2:1108 (Nov. 14) 1931.

CASE DISCUSSION
(Abdominal Pain of Eight Hours' Duration).

J. MADISON GREER, M. D.
FRANK J. MILLOY, M. D.
Phoenix, Arizona

(Case No. 16381, Case Records of the Massachusetts General Hospital, New Eng. Jour. of Med., Sept. 18, 1930, page 592.)

Case History

This patient was an American spinster seventy-four years old who entered the Emergency Ward on June 21 complaining of lower abdominal pain of eight hours' duration.

She gave a history of rather atypical anginal attacks consisting of a sense of constriction and suffocation in the throat and perhaps some radiation in the arm for the past three years. She was known to have had hypertension for several years, just how long is uncertain.

Dr. Burrage was called in to see her at eleven o'clock the morning of admission and was told that at nine o'clock she had sudden abdominal pain. She had five or six bowel movements, not diarrhea, and vomiting. Since that time the bowels had not moved. At the time he saw her the belly was flat, soft, not tender. The heart rate was rapid, about 100 perhaps. Chest clear. Her niece had also had abdominal pain at the same time, and there was some question as to whether she and her niece had had the same things to eat that night. But it turned out on further investigation that they had not. Acute food poisoning or an atypical coronary attack were the things thought of. Dr. Burrage gave her one-quarter grain morphia at the time and came back to see her at three o'clock in the afternoon. He found her unrelieved by morphia. This time the abdomen was moderately distended, still not particularly tender, chest filling up with rales. The pulse was fairly rapid, about 120, with numerous extrasystoles.

She was sent in to the Emergency Ward, where she showed what has been described with the addition of extreme cyanosis with rapid shallow breathing, blood pressure 190. The blood pressure when she was seen in the morning had been 220. She was admitted to the ward and given morphia to keep her comfortable. During the afternoon the blood pressure gradually fell to 110 by evening. She had no more bowel movements. She complained almost continually of pain in spite of one-quarter grain doses of morphia every three hours, enough morphia to bring the respirations down to 15 by evening. The abdomen became more and more distended. The pulse came down to 100 and did not seem so irregular.

The following morning she seemed better so far as the general condition went. She was better able to talk, was not so cyanotic, pulse fairly regular at 100. But the abdomen was tremendously distended and very tender; whenever we touched her or made a motion as though going to touch her she screamed. The leukocyte count was 30,000 the red count 7,500,000. During the day she grew steadily worse. The blood pressure gradually fell so that by evening it was 90 to 100. She had had a great deal of morphia, so that the respirations got down to 10 or 11. The abdomen became more distended. She had no bowel movements. During the evening she began to have fecal vomiting. The pulse went up to 120 or 130, with more or less irregularity. Apparently the chest was clear. At this time as at entry we were not able to examine her back. She died at six o'clock in the morning of June 23, the second day after entry. The leukocyte count had increased to 36,000 at that time.

Discussion by Dr. Greer

The outstanding points in this case are the following:

Aged single woman with acute severe lower abdominal pain, coming on suddenly followed by vomiting, a few bowel movements at first but no more; at first the abdomen was flat, soft and not tender, later it was distended and very tender. The vomiting continued and was fecal in character within twenty-four hours. Condition became rapidly worse with failing heart and disturbed respiration, death occurring within seventy-two hours after the onset. The above picture was associated with some heart signs as increased rate, later decreasing, irregularity, a decreasing blood pressure from 220 to 90, syanosis, rapid shallow breathing a rather unusual blood picture, both red and white count being high. Such a patient ill enough to die within three days with almost no history of past illness makes a very interesting case for speculation.

In a case of this kind we naturally think first of the acute abdominal conditions and endeavor to prove them or rule them out. Some of the conditions we might consider are the following: 1. Acute appendicitis, 2. Acute enterocolitis, 3. Acute salpingitis, 4. Perforated gastric or duodenal ulcer, 5. Acute cholecystitis. 6. Acute intestinal obstruction, 7. Acute pancreatitis, 8. Acute pyelitis, 9. Renal colic, 10. Gastric crises, 11. Acute abdominal allergy, 12. Mesenteric thrombosis.

In consideration of the above conditions we might discuss them briefly, in the light of what is given us in this case, as follows:

Appendicitis can almost be ruled out in a patient of this age from the history that the pain was in the lower abdomen from the first, the vomiting continued through from the beginning, the pain was never localized, there was no rigidity and the tenderness came on later. As to enterocolitis we would expect the pain to be more general, not so severe, more colicky in nature and to have continued diarrhea; nor would we expect it to be severe enough to take the patient's life in so sshort a time. Salpingitis can be ruled out by the negative past history as well as the severity of the condition, with its sudden onset and no mention of pelvic signs or

(Continued on page 92)

Southwestern Medicine

Printed by THE A. C. TAYLOR PRINTING CO., Phoenix, Arizona
Published monthly for the Board of Managers of the four constituent societies.

Volume XVI | February, 1932 | No. 2

EDITORIAL STAF

DR. W. WARNER WATKINS, Box 1587, Phoenix, Arizona.....Editor in Chief
DR. JAMES J. GORMAN, El Paso, Texas.....Associate Editor
DR. ORVILLE H. BROWN, Phoenix, Arizona.....Associate Editor
DR. L. B. COHENOUR, Albuquerque, N. M.....Associate Editor

BOARD OF MANAGERS
ARIZONA STATE MEDICAL ASSOCIATION

DR. D. F. HARBRIDGE.....Phoenix
DR. H. A. REESE.....YUMA

EL PASO COUNTY MEDICAL SOCIETY

DR. PAUL GALLAGHER.....El Paso
DR. W. L. BROWN.....El Paso

NEW MEXICO STATE MEDICAL SOCIETY

DR. C. F. BEESON.....Roswell
DR. H. A. MILLER.....Clovis

MEDICAL & SURGICAL ASSOCIATION OF THE SOUTHWEST
(Publication Committee)

DR. WILLIS W. WAITE.....El Paso
DR. E. PAYNE PALMER.....Phoenix
DR. P. G. CORNISH, Jr.....Albuquerque

SWEET SIXTEEN

Pressure of more important affairs prevented our giving ourselves the customary pat on the back last month, as we started the sixteenth year for SOUTHWESTERN MEDICINE. The editorial office is allowed this self congratulation, usually the only commendation of any sort received during the year. We receive plenty of bricks, but no flowers, so that we may be excused if we take the bricks and erect a playhouse out of them.

The medical profession in the southwest has been in the doldrums during the past year, but that is no occasion for allowing our medical craft to deteriorate. A ship in the doldrums will have all hands at work, painting, scraping, mending, and getting ready for the fair wind. This is a time when we should put our best foot foremost; when we should have the best possible annual meetings; when we should take postgraduate work; when we should read and study; when we should, in every possible way, prepare to do the best work we are capable of in the future. If we have fewer patients, we should give each of them the benefit of additional time and study, of more carefully taken histories, of more exhaustive examinations, of more personally directed treatment. Thus will we find ourselves well in advance of our time, when fair winds come again. So far as we have observed, this is the spirit of the medical profession in the southwest. At least, those who possess this spirit and exercise it NOW, are going to be the leaders of medical thought and activity five years hence.

ARIZONA STATE BOARD OF MEDICAL EXAMINERS

The Secretary of the Board, Dr. B. M. Berger, reports that, at the January, 1932, meeting of the Board, two applicants were licensed by examination and three by reciprocity, as follows:

DR. D. J. LYONS, graduate of St. Louis University in 1930, was licensed by examination; he is located at Yuma, Ariz.

DR. JOHN W. PENNINGTON, graduate of University of Iowa in 1929, was licensed by examination; he is located in Phoenix, practicing urology in association with Dr. David M. Davis, office in the Professional Building.

DR. TREVOR G. BROWNE, graduate of McGill University in 1922, was licensed by reciprocity with Michigan. He is located in Phoenix, offices in the Professional Building, where he practices pediatrics.

DR. WINFRED L. REID, graduate of Northwestern University in 1924, licensed by reciprocity with Mississippi. He is located in the Professional Building, associated with Dr. Orville H. Brown, practicing surgery.

DR. WARREN S. BALDWIN, graduate of Vanderbilt University in 1892, licensed by reciprocity with Oklahoma; he is located at Mayer, Ariz.

Dr. Berger also announces the appointment of DR. WILLIAM G. SHULTZ of Tucson, as a member of the Board, to succeed Dr. A. L. Gustetter of Nogales.

WILLIAM CLYDE ELLIS
W. H. ANDERSON
J. O. HATCHER
G. S. LABEN
E. B. SHAW

The Medical and Surgical Association of the Southwest have lost several valuable and esteemed members by death during the past year. The Association feels the loss of these men very keenly. Their pioneer work in the southwest stands as an inspiration to the younger members of the profession and has

done much to elevate the standard of medical practice in their communities:

Dr. William Clyde Ellis, Phoenix, Ariz., died July 1st, 1931. Dr. Ellis was born at Portsmouth, Ohio. He received his early education in the schools at that place. He was a graduate of the Medical College of Ohio at Cincinnati in the class of 1899. He practiced medicine in Ohio for several years, but in 1907 for climatic reasons he removed to Phoenix, where he acquired an extensive practice, and became one of the better known physicians of the state. He was active in advancing the welfare of the city and valley, and served a term as a city commissioner. He erected the Ellis and Physicians' Buildings, besides other down-town property. He is survived by his wife and a daughter. Dr. Ellis died of carcinoma of the larynx.

Dr. W. H. Anderson, of El Paso, was a prominent physician of that city for many years. His most outstanding work was done in the role of public health officer and it was under his administration that many strides in the supervision of community health were made. His was the chief influence responsible for obtaining certified milk in the community. He died of heart disease at the age of sixty.

Dr. J. O. Hatcher, of Deming, New Mexico, was a very high type of general practitioner and a great loss to the community. A peculiar circumstance in his death is that grandfather, father and son all died at the age of 52. He is survived by his wife and two children.

Dr. G. S. Laben, of Sacaton Indian Reservation, Arizona, was in the government service. He was a faithful attendant of medical meetings and his familiar face will be greatly missed.

Dr. E. B. SHAW, of Las Vegas, N. M., was known as an active and successful medical man whose long experience in practice did not dull his interest in the progress of the profession. Dr. Brown speaks of his surprising vitality and vigor when he last saw him at the New Mexico State Society meeting in 1930 at which time he was nearly eighty years old.

Be it resolved that the sympathy of the Association be extended to the families of the deceased. Be it further resolved that a copy be sent to the members of their families and that this resolution be spread upon the minutes of the society.

HOWELL RANDOLPH,
RALPH HOMAN,
W. A. GEKLER,
Committee on Necrology.

RADIOSENSITIVE TUMORS

(Continued from page 85)

lowing chart, grading from the most sensitive to the least sensitive.

Sensitivity of Normal Tissues.

White Blood Cells	2.5
Germinal Cells of Ovary and Testis	2.25
Blood Forming Organs, spleen and marrow	2.
Endocrines, thyroid, adrenal, pituitary	1.8 to 1.6
Blood Vessel Intima	1.5
Hair Follicle	1.4
SKIN,	1.
Serous Membranes	.9
Intestines	.8
Liver and Pancreas	.7
Uterus and Kidney	.6
Fibrous Tissue	.5
Muscle	.4
Bone	.3
Nerve Tissue	.2
Fat	.1

It will immediately be apparent why we can get such prompt effect on the white blood cells and on the germinal cells of ovary or testis without injuring the skin, or why we can destroy an angioma beneath the skin by producing an obliterating endarteritis through effect on the blood vessel intima, and, on the other hand, why it is so difficult to get effect on an osteogenic sarcoma without injuring the skin.

Bergonie and Tribondeau, about 1915, formulated the law that radiosensitivity is in direct ratio to the rapidity of cell growth and the approach to the embryonal type of cell. This law still holds, though we have gradually come to recognize the place which various other factors play in sensitivity. Some of these can be briefly mentioned:

Age of patient has an influence; cells are more sensitive in the child and become less sensitive with increasing age.

Blood supply; anemia decreases the sensitivity while engorgement increases it; a hyperemic skin will stand only half the radiation dose that a blanched skin will stand.

Temperature; heat increases the sensitivity and cold decreases it, this probably being due to the difference in vascularity.

Structure; the more cellular the structure the more sensitive it is. Actively dividing cells or those with active metabolism are decidedly more sensitive than those in a resting stage; cells with secretory function are particularly sensitive in the stage of metabolic activity.

Systemic disease; syphilis increases cell sensitivity. Anything which diminishes the resistance of the cell makes it more sensitive to radiation. Chemicals increase the effect of radiation, particularly inorganic compounds.

For abnormal growths, the general physiology of the cell of origin determines the sensitivity of the growth to a large extent.

Coming to abnormal tissues, such as tumors, we find that the radiosensitivity is in proportion to the rapidity of metabolism and of growth, with due regard for the cells of origin. The reasons for the sensitivity are briefly (1) cells are more vulnerable when the nuclei are in mitoses, (2) the cell is more embryonal in type when multiplying rapidly, (3) the unstable vascularity and poorly distributed nourishment, (4) cells are anaplastic, without developed function.

The classification of tumors according to sensitivity would be about as follows:

Most Susceptible: Lymphomas, lymphosarcoma, leukemia, Hodgkin's thymic tumors, myeloma.

Very Susceptible: Embryonal tumors and cellular anaplastic tumors, such as carcinoma of testis or

Pulvules Sodium Amytal

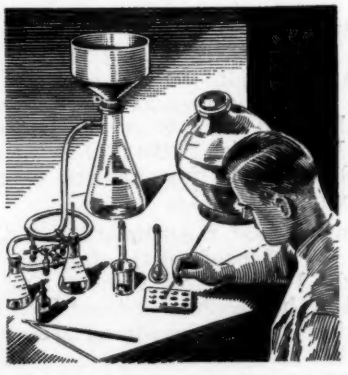
IN SURGERY

FOLLOWING the administration of Pulvules Sodium Amytal (*Sodium Iso-Amyl Ethyl Barbiturate*) tranquillity replaces preoperative anxiety and excitement; less anesthetic is required; postoperative nausea is absent or diminished. Pulvules Sodium Amytal are distinctly useful in surgery, in obstetrics, and in general practice.

Supplied through the drug trade. Write for literature.

Eli Lilly and Company

INDIANAPOLIS, U. S. A.



Determining the Hydrogen Ion Concentration of Sodium Amytal Solution Colorimetrically

ovary, carcinoma of breast, stomach, esophagus and anaplastic sarcomas.

Susceptible: Basal cell carcinoma.

Resistant: Adenocarcinoma and adenoma; desmoplastic tumors like squamous carcinoma.

Very Resistant: Fibroblastic sarcoma, osteosarcoma, neurosarcoma, glioma.

The so-called grading of tumors is based on the degree of anaplasia and the approach to the embryonal type of cell. The higher the degree of anaplasia, which means the absence of functional differentiation of cells, the more sensitive they are to radiation. The rapidity of response to radiation follows more or less closely the sensitivity and is well borne out in these two cases. It is interesting to get a bird's eye view of the rate at which various lesions respond to radiation treatment. Hirsch groups them into four classes.

RESPONSE IN ONE DAY TO ONE WEEK

Lymphosarcoma
Leukemia
Hodgkin's granuloma
Mycosis fungoides
Psoriasis (early)
Eczema
Lichen ruber
Epidermophyton.

RESPONSE IN ONE TO TWO WEEKS

Round cell sarcoma
Prostatic hypertrophy
Tuberculous granuloma
Tuberculosis of bone
Fibroadenoma
Lupus hypertrophicus
Favus and Sycosis
Acne vulgaris
Verrucae juvenilis.

TWO TO THREE WEEKS

Epithelioma
Carcinoma
Melanosarcoma
Angiosarcoma
Acne vulgaris
Lupus planus
Goitre.

THREE OR MORE WEEKS

Keloid
Fibrosarcoma
Chondrosarcoma
Fibroma
Fibromyoma
Osteosarcoma
Osteoma
Neuroma
Verrucae.

In the case of the man with the mediastinal growth, the rapidity of response was quite spectacular, almost like a dose of adrenalin in asthma. In one week's time the large tumor shown which filled the entire mediastinum and pressed upon the trachea and great blood vessels, had entirely disappeared. There is sufficient gradation in response of lymphosarcoma, Hodgkin's, tuberculous adenopathy and carcinomatous metastases in glands,—to radiation,—that the method can be used in differentiating. The response of this growth would throw it in the lymphosarcoma group, though not differentiating it from leukemia.

In the girl, the tumor mass in the abdomen began to shrink after the second treatment, or within one week. The shadow in the left chest showed marked change within a four day period. This is almost enough to establish a diagnosis of lymphosarcoma, also. To my mind, it would rule out hypernephroma.

DR. D. M. DAVIS questioned the accuracy of the conclusion that this could not have been a hyper-

"WHAT A PLEASANT WAY TO GET BACK MY STRENGTH, DOCTOR"

Extra nourishment

without extra burden on the digestion

DOCTORS often say that, in convalescence, one of the greatest problems is nutrition. Yet it is as frequently necessary to persuade the patient as it is to fight the condition.

High nutritive value, united with quick digestibility and assimilation, at once suggests milk. But many patients dislike milk. Even those who detest plain milk, or who have no appetite, yield to the delicious chocolate flavor of Cocomalt.

Far more important, however, Cocomalt actually adds 70% to the nutritive value of every glass or cup of milk. For Cocomalt is a scientifically balanced combination of milk proteins, milk minerals, cocoa, eggs, barley malt and sugar.

It is for these cogent reasons that physicians are increasingly recommending Cocomalt in convalescence, in general debility, for expectant and nursing mothers, and for growing children.

Cocomalt is sold in both grocery and drug stores in ½ lb., 1 lb. and 5 lb. cans. In powder form, ready for instant mixing with milk.

Free to Physicians

We should be glad to send you a trial can of Cocomalt for testing. Simply use coupon.

Cocomalt

DELICIOUS HOT OR COLD

ADDS 70% MORE NOURISHMENT TO MILK

R. B. DAVIS CO., Dept. 632 Hoboken, N. J.
Please send me, without charge, a trial can of Cocomalt.

Name.....

Address.....

City..... *State*.....

nephroma because of the rapidity of change after radiation, citing a case where a similar tumor with lung metastases in a young girl had undergone a very similar rapid disappearance following radiation. Nephrectomy was then performed and the histologic findings proved conclusively that it was hypernephroma, the patient dying about nine months later from metastatic extensions. He did not think that one could depend on the response to radiation in making differentiation between tumor growths. He also raised question as to the response of prostatic hypertrophy to radiation.

CASE DISCUSSION

(Continued from page 87)

symptoms. In perforating gastric or duodenal ulcer we would expect sudden pain but it should be in the upper abdomen and there should be tenderness and marked rigidity, board-like rigidity of the upper abdomen, as well as previous history of gastric disturbance, although this is not always obtainable. In acute cholecystitis we would expect the signs and symptoms to be at least directed toward the upper right quadrant as well as some previous history of digestive or gall bladder symptoms. As to acute intestinal obstruction, it is quite evident that this patient had acute intestinal obstruction from the sudden onset, from the lack of tenderness and distention at first and from the vomiting which continued and gradually became fecal a short time before death. This condition will be considered again later. In acute pancreatitis, the pain is in the upper abdomen, the condition does occur in the aged and it is an abdominal calamity accompanied with serious and severe symptoms and shock, and the patient may die in a very short time. There is usually severe tenderness and rigidity from the first, with the signs and symptoms confined, at first at least, to the upper abdomen. In cases of acute pyelitis, the symptoms usually do not come on quite so suddenly and the signs and symptoms are directed toward one or the other of the two sides and toward the back. There should be chills and fever and tenderness in the lumbar region. Nothing is said about the urine in this case, so we take it that it was negative. Urinalysis would help in ruling out an acute pyelitis, as well as renal colic. Gastric crises can be ruled out as we have no history or blood picture or pain in the gastric region to make us think of such a condition. Acute abdominal allergy can probably be ruled out because of the severity of the case. It is possible that we should consider this condition and it is noted that acute food poisoning was considered and is mentioned but given up apparently. In this condition we get diffuse abdominal pain of a colicky nature with distention, nausea, and vomiting, and the pain is usually most marked in the lower abdomen. I suppose it would be possible to get edema enough with this condition, especially in a patient of this age, to start off an intussusception or volvulus which would explain the symptoms and signs. However, acute abdominal allergy is quite rare and I should not like to say that it was the cause in this case. Mesenteric thrombosis cannot be ruled out, and the age of the patient, the history, the hypertension, the anginal attacks, which in all probability were coronary sclerosis, point toward a condition of this nature.

In going over this case we cannot rule out intestinal obstruction and in fact we feel quite sure that the condition is one of acute intestinal obstruction. The cause of the obstruction, in the absence of a history of previous surgery, or abdominal or pelvic inflammation of any kind, is not quite certain. However, in considering the age of the patient, the brief and scanty history, the mention of hypertension one would think of a mesenteric thrombosis as a cause of the obstruction. One should not forget, however, that in a patient of this age it is possible for an obscure and latent tumor of the intestinal tract to start off a volvulus or an intussusception. In considering a mesenteric thrombosis one would expect to get blood in the stool; but inasmuch as this patient had no stools after the first few at the beginning of the attack it is very possible that it would not be detected. As to the blood picture, a white count of 30,000 is in keeping with mesenteric thrombosis according to Graham. I am at a loss to explain the high red count unless it might be due to a decrease in the fluid content of the blood and this count a relative one.

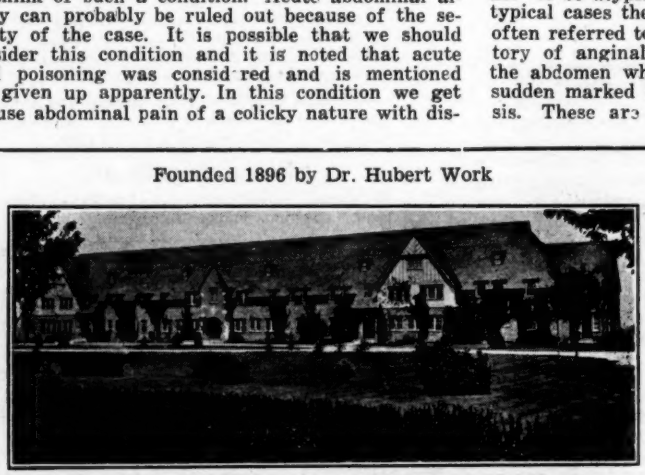
After this brief consideration and discussion of the case I should like to venture a diagnosis of:

1. Acute intestinal obstruction due to mesenteric thrombosis probably due to arteriosclerosis of the superior mesenteric artery.
2. General arteriosclerosis with hypertension.

Discussion by Dr. Milloy

The question immediately presents itself in this case, as it does in all such cases in old people, where there is severe abdominal pain, to distinguish between the pain of coronary occlusion and the pain of true abdominal disease. In older people, any of the acute abdominal conditions are just about as liable to be atypical as they are typical. And in less typical cases the pain of coronary occlusion is very often referred to the abdomen. Here we have a history of anginal attacks, a severe pain located in the abdomen which is not relieved by morphine, a sudden marked fall in blood pressure, a leukocytosis. These are pathognomonic symptoms of coro-

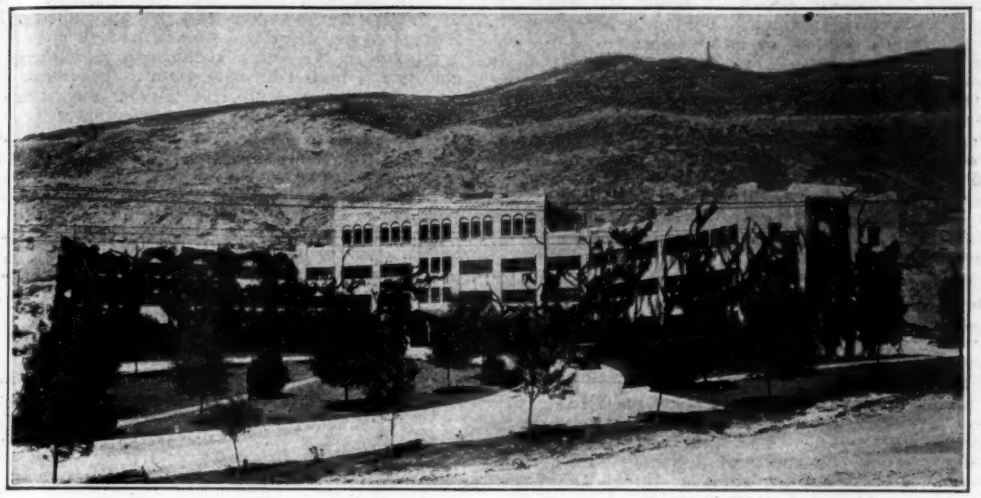
Founded 1896 by Dr. Hubert Work



A modern, newly constructed sanitarium for the scientific care and treatment of those nervously and mentally ill, the senile and drug addicts.

CRUM EPLER, M. D.
Superintendent

WOODCROFT HOSPITAL, PUEBLO, COLORADO



THE HOMAN SANATORIUM

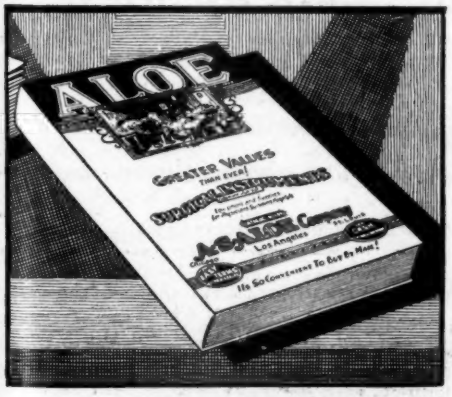
For the Treatment of Tuberculosis

EL PASO, TEXAS

A privately owned, thoroughly and modernly equipped, ethical institution of 110 rooms. Heliotherapy, pneumothorax, phrenic nerve and chest surgery, and all other modern methods of treatment.

Write for booklet giving rates and other information.

Doctor! Send for this 416-Page Catalog



of

Surgical Instruments, Equipment and Supplies

Contains listings of your every want at money-saving prices. Thousands of items: "White Steel" Furniture, Reception Room Furniture, Physical Therapy Apparatus, Chrome Plated Instruments, Diagnostic Instruments and Apparatus, Laboratory Apparatus, Supplies, Orthopedic Supplies, Etc. Everything for a distinctive armamentarium. Just mail us your name and address. It's FREE. No obligations.

Just Mail Your Name and Address---It's FREE!

A. S. ALOE COMPANY

932 South Hill Street,
LOS ANGELES, CALIFORNIA

nary occlusion with cardiac infarction. And this is exactly the type of patient in whom you would expect a coronary occlusion, having a hypertension, which, at the age of 74, practically means arteriosclerosis. However, by late afternoon she began to develop abdominal distention and by the following morning the case was decidedly one of abdominal disease, as all the chest symptoms had subsided. The gradually increasing abdominal tenderness and marked abdominal distention, the leukocyte count of 30,000, along with the symptoms of shock, such as cyanosis, rapid shallow breathing, marked fall in blood pressure, rapidly increasing pulse, and finally fecal vomiting, mean general peritonitis, or intestinal obstruction.

The history is not very suggestive of an obstruction. Obstruction is not liable to be preceded by loose bowel movements, and it is unlikely that she would have 30,000 leukocyte count so soon. And, while she vomited at the very start, she did not vomit again until near death, at which time it really was fecal vomiting. It is much more likely that she developed general peritonitis from rupture of some hollow viscus. The most common things to suspect would be ruptured appendix or ruptured peptic ulcer. The less common things to suspect would be a ruptured gangrenous gall bladder, rupture of an intestinal ulcer, e. g., an amebic ulcer or an ulcer of ulcerative colitis. There is absence of any history to suggest either gall bladder or ulcer, and besides pain was in the lower abdomen. A sudden severe pain in the abdomen with symptoms of shock and prostration, is exactly what happens when a peptic ulcer ruptures. It is not likely that such a leukocytosis would develop so rapidly, but of course it could produce a general peritonitis which, of course, would cause a high leukocyte count. Diarrhea, or loose bowel movements, frequently precedes appendicitis. She may have had some indigestion and taken a laxative. One of the best things to precipitate a severe case of appendicitis is a strong physic. Here is a woman who developed sudden pain in the lower abdomen and vomited; she also has a high leukocyte count. The most likely thing is acute appendicitis. Desver says the commonest cause of belly ache is appendicitis. It is not unusual in old people to have acute appendicitis, and to have no pain or tenderness over McBurney's point. She probably had no pain until the appendix ruptured and still did not have tenderness until the peritonitis developed. There is no way to diagnose ruptured intestinal ulcers unless we have a history of the primary cause of the ulcer.

Diagnosis: Appendicitis, ruptured, and general peritonitis.

Autopsy Findings
Pathologic Discussion

Mitral stenosis.
Arteriosclerosis of the aorta and the great vessels.
Slight arteriosclerosis of the coronary arteries.
Hypertrophy of the heart.
Embolism and thrombosis of the superior mesenteric artery.

Dr. Merrill J. King: At autopsy this patient was found to have a moderately enlarged heart with definite mitral disease. The mitral orifice was practically occluded, and there were old healed vegetations on the leaflets. Both cusps were sclerosed and calcified. In the posterior cusps about 7 millimeters from the free margin there was an irregular narrow, eroded, slit-like area of destruction forming an opening about one centimeter long and from two to four millimeters in width. The edges of this perforation were finely nodular. There were two small finely granular areas on the auricular surface of the right anterior cusp. There was slight

An Invitation To the Doctors of the South

THE DALLAS SOUTHERN CLINICAL SOCIETY

Will hold its Fourth Annual Spring Clinical Conference in Dallas
March 28, 29, 30, 31, April 1, 2, 1932

20 DISTINGUISHED GUEST SPEAKERS IN:—

General Assemblies:—
Five 45 minute addresses each morning on topics of general medical interest.

Round Table Luncheons—
On five days in groups:—Medicine, Surgery, E. E. N. T., Pediatrics, Urology, and Orthopedics.

Hospital Clinics:—
On four afternoons at six hospitals. Forty practical demonstration clinics.

ELABORATE EVENING ENTERTAINMENTS:

Monday—General Public Meeting.
Tuesday—Symposium on "Biliary Tract Disease" by three distinguished clinics.
Wednesday—Brilliant Clinic Dinner.
Thursday—Symposium on "Arthritis."
Symposium on "Conservation of Maternal Health."

NINETY-SIX ONE-HOUR PERIODS OF POST-GRADUATE INSTRUCTION:—

Arranged in coordinated courses to prevent conflicts with hospital clinics.

POST-CONFERENCE ACTIVITIES:—

Friday afternoon and Saturday morning. Medical ward-rounds and surgical operative clinics by Dallas members.

ALL-INCLUSIVE REGISTRATION FEE—$10.00.

For detailed information:
Consult your Prospectus, or address

DR. M. O. ROUSE, Secretary,
1424 Medical Arts Building,
Dallas, Texas.

SIXTEENTH ANNUAL CLINICAL SESSION

AMERICAN COLLEGE OF PHYSICIANS

April 4-8, 1932

San Francisco, Calif.

Outline of Session

TIME	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY
	April 4	April 5	April 6	April 7	April 8
9:00 A. M. to 12:00 M	Morning free. Registration, exhibits, etc.	1st Clinical Session	2nd Clinical Session	3rd Clinical Session	4th Clinical Session
12:00 M to 2:00 P. M.	Luncheon	Luncheon	Luncheon	Luncheon	Luncheon
2:00 P. M. to 5:30 P. M.	1st General Session	3rd General Session	5th General Session	6th General Session Annual Business Meeting	7th General Session
5:30 P. M. to 8:00 P. M.	Dinner	Dinner	Dinner	ANNUAL BANQUET	Dinner
8:00 P. M. to 10:30 P. M.	2nd General Session	4th General Session	Convocation and Reception to New Members		"'49 Party"

A POSTGRADUATE WEEK IN INTERNAL MEDICINE AND ASSOCIATED SPECIALTIES, covering a wide range of subjects presented by outstanding men in medicine not only from the west coast, but from all parts of the country. About half of the program will be devoted to actual clinics, laboratory demonstrations, ward walks and exhibits. In addition, there will be complete displays of medical literature, pharmaceutical products, equipment, appliances, special foods, etc.

Transportation—Railroad fares will be reduced to one and one-half times the one-way fare for the return trip. A "certificate of identification" must be secured from the Executive Secretary of the College to entitle physicians and members of their immediate families to these reduced fares.

San Francisco Headquarters—Palace Hotel, New Montgomery and Market Streets.

Post-Convention Tour—Following the San Francisco Session, a special train has been engaged for a tour of the Southwest, including the Yosemite Valley, southern California (with a two-day program of pleasure and inspection around and in Los Angeles), the Grand Canyon of Arizona, etc. Special itinerary and rates sent upon request. Los Angeles headquarters will be the Los Angeles Biltmore Hotel.

INVITATION extended to all qualified physicians. Non-members of the College will pay a nominal registration fee.

Address inquiries and requests for programs to the Executive Secretary.

S. Marx White, M. D., President
Minneapolis, Minn.

Wm. J. Kerr, M. D., General Chairman
San Francisco, Calif.

E. R. Loveland, Executive Secretary
133-135 S. 36th Street
Philadelphia, Pa.

sclerosis of the coronaries and aorta, with a large calcified plaque in the arch.

The next most interesting findings were in the superior mesenteric artery, where at the origin of the first large branch supplying the jejunum there was a small firm dark red blood clot 1.5 centimeters in length resting against the angle formed by the bifurcation of the vessel. This mass was slightly adherent to the vessel wall and practically occluded the lumina of both the main vessel and the branch. Practically all of the jejunum and portions of the ileum and the ascending colon were dark red and distended and were beginning to undergo infarction. There was about 400 cubic centimeters of serosanguineous fluid in the peritoneal cavity, with fresh fibrinous adhesions between contiguous loops of gut.

The liver was negative. The kidneys showed arteriosclerotic changes.

Dr. William D. Smith: There are two or three rather interesting things about this case. When I saw the patient the morning after she came in it was perfectly obvious that she had some abdominal tragedy, that is perforation, peritonitis or thrombosis. The diagnosis we made of thrombosis of course was guesswork. At her age, with her heart and the tremendous suddenness of the thing it seemed to me the best probability. It happened to be right; but there might perfectly well have been some other abdominal condition.

Another interesting thing was the drop of 100 points in blood pressure in spite of the shock. The heart rate was about 100 and the sounds were fairly good. That seemed a little queer with the lungs full of rales. Even so when we listened to her heart it was a pretty good heart. Why she did not have a rate of 160 I cannot imagine.

The question of cononary thrombosis was brought up on admission. I do not know why she might not have had coronary thrombosis. I have seen the pain of coronary thrombosis maximum in the back, I have seen it maximum in the neck and teeth and I have seen it imitate gall bladder very closely; but I have never seen the pain of coronary thrombosis in the lower abdomen.

She was seventy-four years old and had a practically occluded mitral valve and no one raised the slightest question of mitral stenosis. There was no history of congestive failure, the heart was not fibrillating, there was no murmur suggestive of mitral stenosis. Once in a while you see an old person with no past history of rheumatism who has definite mitral stenosis, and you wonder if a person can have mitral stenosis from an unknown rheumatic infection in childhood, get to be seventy-five or eighty years old with no heart symptoms, then die suddenly and show practical occlusion of the mitral valve. I have always wondered whether there might not be arteriosclerotic mitral stenosis just as there is arteriosclerotic aortic stenosis.

Dr. Edward L. Young, Jr.: Surgically I do not think there is anything to add. This case is of the type in which surgery offers nothing. As a matter of fact the surgical mortality in mesenteric thrombosis is very high anyway. The only figures I know put it at 94 per cent. In a person of this general type that does not leave any loophole at all. That seemed to be perfectly obvious when I saw her. We did not see any way of making a diagnosis between mesenteric thrombosis or embolus and perforation with peritonitis. The background of heart disease and arteriosclerosis made the other the first choice. I do not know—perhaps Dr. Richardson can tell—whether any case has been operated upon successfully in this hospital. I heard Dr. Balch speak of one in which he and Dr. Porter took out about four feet of intestine with success. The one or two cases I have done have not been successful. Medically I do not know that the mortality is any better. It is an interesting fact, however, that there have been reported one or two cases of autopsy with more or less complete occlusion of mesenteric vessels yet the patient not dying from that condition. Those are cases of slow onset, not this clinical type of sudden onset.

Dr. Smith: I have wondered whether people ever have minor mesenteric thrombosis and get over it. I remember one patient who had cerebral embolus, emboli in both lower extremities and then suddenly had an attack of intense abdominal pain. We decided she had mesenteric embolus with thrombosis. Then she proceeded to get over it. The diagnosis of course was up in the air. I do not think she had gall bladder disease. She had had three emboli already.

Dr. Edward P. Richardson: I know that the mortality is very high in mesenteric embolism and thrombosis. One case recovered after extensive resection between 1908 and 1918. I cannot say since then. But at any rate, with any extensive lesion the only chance is resection of the affected intestine. I should say it is possible to have embolism to such a limited extent that it might not permanently interfere with the circulation of the bowel. One of the striking things in these cases is the leukocyte count, which is often higher than is present in infection. Another source of difficulty is that interference with bowel circulation is sometimes due to embolism and sometimes to thrombosis. In embolism there is ordinarily quite clear-cut demonstrable pain. In thrombosis there is often a very indefinite history, and the condition may be found at autopsy when quite unsuspected.

CASE DISCUSSION
(An Acute Abdomen in Child)

F. C. JORDAN, M. D.
HOWELL RANDOLPH, M. D.
Phoenix, Arizona

(Case 11053, Case Records of Massachusetts General Hospital, from Boston M. & S. Jour., Jan. 29, 1925, page 229).

Case History

A six-year-old schoolgirl came to the Emergency Ward September 10. She had had sore throat, fever, frequency and pain in the right side for the past few days.

F. H. Good.

P. H. She was born at full term, normal delivery, and weighed only about four pounds at birth. She was breast fed until one year old, grew well and developed normally. She had measles, mumps, chickenpox and whooping cough between two and four years without serious complications. The winter before admission she had a swollen, red and tender kneejoint for two weeks and was thought to have rheumatic fever.

P. I. September 5 she complained of sore throat and was feverish and restless. The following night she vomited greenish-yellow fluid several times and complained of epigastric pain. September 6 she felt better, but could not retain food or fluid taken by mouth. September 7 she complained of the pain's being in the right side. September 9 she was so tender that she did not want to be lifted out of bed. Her bowels, which usually tended to be constipated, moved well September 9 after a cathartic. Since the onset she had urinated almost every hour by day and frequently at night. There had been no scalding.

P. E. A fairly well developed and nourished child looking rather ill, but mentally alert and cooperative. Face pinched. Eyes slightly sunken. Conjunctivae

injected. Head negative. Cervical glands slightly enlarged. Chest long and narrow. Lungs and heart normal except for rapid heart action. Abdomen full and tympanitic. Slight tenderness on deep pressure over right lower quadrant. Marked tenderness over the right costovertebral area. Less tenderness in the right flank. Spasm not marked. Extremities, pupils and reflexes normal. Genitals not recorded.

Temp. 99 to 104, pulse 110 to 150. Before operation R. 20-36, amount of urine not recorded, sp. gr. 1.006-1.012, neutral at two of five examinations, no albumin or sugar (a catheter specimen was taken); blood, hgb. 100%, leucocytes 15,000-23,400, polynuclears 84%, reds normal. X-ray negative. First surgical consultant: (1) Spasm most intense in costovertebral region. (2) Rectal examination negative. (3) Temperature 105°. This suggests kidney rather than appendix. Ssecond surgical consultant: Probable pyelitis. Genito-urinary consultant: "The tenderness is definite to-night (September 12). It seems confined to the right upper quadrant and the right side of the abdomen and is most definite in the costovertebral angle. . . . . Recommend operation."

September 13 there was less tenderness in the costovertebral angle but much in the right flank; no spasm or tenderness over the appendix.

September 15 operation was done. Seven hours after it the temperature suddenly rose to 104.6°, the pulse to 170, and the respirations to 50. There was generalized tendernes and spasm over the abdomen. The chest was clear. The next day the patient appeared much worse. Subpectoral saline 1000 c.c. was given in the morning and at night. Shortly after midnight the child died.

Discussion by Dr. Jordan

I believe we can say that we have one of three things: (1) appendicitis with a retrocecal abscess; (2) a subphrenic abscess; (3) abscess within or around the right kidney. What evidences have we for or against these diagnoses?

Can we eliminate appendicitis? Appendicitis is not rare in a child at the age of six and is usually diagnosed after the appendix is ruptured and there is localized or general peritonitis. The child had some pain and tenderness in the right side, a fever and a leukocytosis of 15,000 to 23,000. The lungs were normal although no x-ray pictures were taken. These are the typical findings of an appendicitis except that you would expect more tenderness and rigidity in the right side. Against the diagnosis of appendicitis, there was a fever of 104 and 105. Such high temperatures are uncommon in appendicitis. Rectal examination also was negative and, in appendicitis, you would expect quite a marked tenderness in the right side on rectal examination; then such a marked tenderness and spasm over the right costovertebral area would not be expected in appendicitis.

How about a subphrenic abscess? This could account for all the signs present. X-ray of the piaphragm on the right side will often determine the absence or presence of the most important sign of the subphrenic abscess. The diaphragm is usually raised and the liver pushed down. Yet, without this evidence, we cannot rule out a subphrenic abscess either secondary to a perinephritic abscess or to an appendicitis.

What evidences have we for or against a pyelonephritis or a perinephritic abscess? One consultant thought that the condition was probably due to a pyelitis. Pyelitis is a term used to designate a mild degree of renal infection. Pyelonephritis is a more extreme grade of infection. This certainly was not

THE ROBINSON CLINIC

Melancholia is a common condition found in adults of all ages. The chief symptom is a feeling of unworthiness and lack of desire to continue with the struggles and problems of life. Women, at the menopaus, frequently resurrect some overt act committed early in life and dwell upon this, until it is magnified into an unpardonable sin. They have delusions that other members of their family are being punished for this sin.

Younger adults are seldom affected with the above formed delusions, but have just as great a sense of their own unworthiness, but can seldom give such a clear and lucid account as to the cause.

All forms of melancholias and depressions must be guarded closely against suicide. Their own statements must not be given credence, because their mental reactions change so quickly, they may deny any thought of self-destruction one minute, and the next, be carefully planning to take their own life. Homes and hospitals offer so many opportunities for suicide, we believe the only safe place for these unfortunates is in a hospital with special equipment to handle these cases, and with carefully trained and watchful attendants; they must be constantly supervised.

Treatment consists of removing the cause, in addition to the above supervision. Careful examinations must be done, to determine the cause. Gland extracts, sedatives, hydrotherapy and psycho-therapy and specific treatment produce the best results.

CURTIS-WRIGHT FLYING SERVICE

Nervous and Mental Diseases

G. Wilse Robinson, Jr., M.D.
Associate Medical Director

G. WILSE ROBINSON, M.D.
Medical Director
1432 Professional Building
8100 INDEPENDENCE ROAD
KANSAS CITY, MO.

Drug and Alcohol Addiction

Paul A. Johnson, M.D.
Internist

a mild infection and, if the kidney was involved, it was more than a pyelitis. Pyelitis is much more frequent in females. About 85 per cent of the cases are female. The increased frequency mentioned was probably due to an accompanying cystitis. Hemholtz has produced primary lesions in the renal pelvis of rabbits by the intravenous injections of staphylococcus and streptococcus, but was unable to to produce these lesions with the colon bacillus. Nelson and Schloss believe that there is an acute inflammatory process of the interstitial tissues of the kidney in the cases that are usually determined to be pyelitis. The colon bacillus is found in about 85 per cent of the cases, staphylococcus in 10 per cent and streptococcus and bacillus proteus in 5 per cent of the cases. Infections due to cocci require radical measures, as nephrectomy. Colon infections are not nearly so likely to be fatal and should be treated conservatively. The colon bacilli have a predilection for the renal pelvis and do not give rise to suppuration. The pyogenic group show a predilection for the renal cortex and the lesions consist of perinephritic abscess, cortical abscess and diffuse suppuration. Hunt's review of 106 cases in Mayo's Clinic of perinephritic abscesses, found that 44.3 per cent were due to primary renal disease, pyelonephrosis, lithiasis, tuberculosis, etc., and 55.7 per cent occurred in clinical absence of apparent or demonstrable or primary renal lesions. Also, that a large percentage are metastatic from some previous infection like carbuncle, tonsillitis and wound infection. Only one of these cases was bilateral. It would seem that we have all the clinical signs of a perinephritic abscess in this case. There was a high temperature, a marked leukocytosis, a tenderness in the right costovertebral angle, a history of a previous perinephritic abscess. The prognosis in in subphrenic abscesses is usually grave.

Our diagnosis in this case is: first, perinephritic abscess: second, subphrenic abscess. I do not see how we can rule out either of these diagnoses.

Discussion by Dr. Randolph

The case under consideration is one in which sepsis is unquestionably the outstanding thing and the location of the central focus of infection is the unsolved problem. The possible diagnoses are perinephritic abscess, subphrenic abscess, pyelitis, appendicitis, liver abscess, hepatitis, pancreatitis or a combination of two or more of these conditions. The onset with sore throat and fever would indicate that the abdominal condition was due to metastatic infection from diseased lymphoid tissue in the throat. Any one of the above conditions might result, with the tonsils as the portal of entry. The second day of the disease she could not retain food but did not complain of pain until the third day. The extreme tenderness of the right abdomen and costovertebral angle on the fifth day would indicate rapidly increasing tension in a suppurative process. Frequency of urination since the onset calls attention to the urinary tract, although there was no "scalding" and no pus cells were reported in the urine. Suppurative process in the kidney itself can be present without pus being present in the urine.

Practically all the symptoms and signs are consistent with appendiceal abscess. A small percentage of appendix abscesses point posteriorly forming psoas abscess. A large number result in subphrenic abscess. Pyelitis is ruled out by the absence of urinary findings. With a hepatitis we might expect a more prolonged illness with more evidence of dysfunction of the liver. A localized liver abscess, however, might very well cause this clinical picture. Pain in the back radiating upward would be suggestive. We would expect pain in the back also with a diffuse pancreatitis which also would be apt to run a more chronic course. It seems to me that the diagnosis lies between appendiceal abscess with developing subphrenic extension and perinephritic abscess.

Perinephritic abscess is the result of a primary pyelitis frequently extending through to the perirenal fat capsule. It is not unusual for hematogenous infection to select this site for abscess formation. The course of the disease is usually longer, there is apt to be more superficial tenderness over the costovertebral area and lumbar fixation from associated spasm of iliopsoas and regional muscles is very common. The operation that was performed might well be expected to have brought the temperature down if perinephritic abscess were the correct diagnosis.

Of considerable importance is the finding of abdominal distention, and tympanitis. I am inclined to place much emphasis upon this point together with the initial fairly high temperature with a subsequent drop and after a day or two another rise. This, and the rapidity with which the disease progressed, suggests a general peritonitis. Increasing tenderness in the right upper quadrant was noted two days after admission. No mention is made of the type of respiration although there is some respiratory embarrassment. It is not stated whether sharp pain was caused by deep breathing or whether there was bulging of the lower intercostal spaces.

Diagnosis: Acute appendicitis, general peritonitis, subphrenic abscess.

Operative Record

PRE-OPERATIVE DIAGNOSIS

(1) Perinephric abscess.
(2) Appendix abscess.

Ether. Before operation the patient had a rapid pulse, but the abdomen remained soft and peritonitis was thought not to exist. Through a right loin incision carried down though the muscles the fascial layers and the intramuscular spaces were found to be edematous as in perinephritis. The perirenal fat was edematous, but the kidney was normal. After this exploration was made it was found that an abscess apparently within the abdomen lay in contact with the lower pole of the kidney. The peritoneum was opened without exposing the abdominal cavity. A mass of adhesions seemed to have effectively walled off the abscess cavity from the peritoneum. On opening through the peritoneum about two drams of thick pus was evacuated from an appendix abscess about one inch in diameter. Rubber wicks and rubber tube drains were placed in the cavity, but no attempt was made to find or remove the appendix. The patient was in fair condition at the end of operation.

Post Mortem Findings

1. Primary fatal lesions.

Gangrenous appendix, abscess and general peritonitis.

Dr. Richardson: The abdomen was slightly distended, the wall a little rigid. The peritoneal cavity contained much thin purulent fluid. The peritoneum generally was coated with fibrinopurulent exudate. The proximal half of the appendix showed edema of the wall, but it was free and the mucosa negative. The distal half was closed off in the region of its proximal portion, and in the situation there was a mass of purulent infiltrated adhesions which walled about an abscess cavity containing dirty reddish fibrinopurulent material in the midst of which rested the necrotic remains of the distal half of the appendix. The retroperitoneal tissues, in the region extending up to and involving to some extent the perirenal tissues in the region of the lower pole of the right kidney, showed purulent in-

filtration. On the right posterolateral aspect of the mass there was purulent infiltration and necrosis to which the operation wound led down. Purulent infiltrated adhesions also extended to the lower end of the ileum. The peritoneum in the region was coated with exudate.

The small intestine was slightly distended and contained much dirty reddish opaque fluid material.

The mesenteric and retroperitoneal glands were slightly enlarged and reddened. The liver was at the costal border in the right mammillary line. The diaphragm was at the third space on the right, the fourth rib on the left.

In each pleural cavity there was about 100 c.c. of cloudy fluid and fibrin. The pleura over the lower lobes showed a slight fibrinous coating.

The thymus gland was present, weighed 8 grams, and was negative.

The lung tissue generally was pale red, spongy, and yielded a moderate amount of reddish frothy fluid. In the region of the lower lobes there were a few scattered areas of hemorrhagic edema.

REVIEW

The maginficent rise of medical and dental thoughts and its release from the inhibitions placed upon it by medieval religion and bigotry, to the freedom of thought characteristic of the modern era has received, during the past year, considerable publicity in a form calculated to reach an extremely large number of persons. Interest in this subject, for doctors not already familiar with it, will lie in the relation of radio to the professions; in the passibilities of radio as a medium for acquainting the public more thoroughly with the aims and achievements of medicine and dentistry.

For the period of a year, the Columbia Broadcasting System has carried over a chain of approximately 40 radio stations a series of weekly talks by Howard W. Haggard, M. D., associate professor of applied physiology at Yale University. The title of the series was fixed with a view to attracting the laity: "Devils, Drugs, and Doctors." The material was prepared by Dr. Haggard in a popular style, with the average level of the public in mind. The talks were ethical and dignified at all times.

The material of the series was historical at times, explanatory at times, analytical at other times. Whatever the approach of each weekly talk, the effect, especially the cumulative effect of hearing the talks week after week, can not but have given the radio public greater sympathy with the professions and greater understanding of their achievements, their objectives, their methods.

Typical titles among the 52 fifteen-minutes broadcasts were:

Medicine, and its Place in History
History of Anesthesia
Florence Nightingale
The Importance of Diagnosis
Mothers' Day?
National Hospital Day
The Black Death
Wilhelm Konrad Roentgen
The Progress of Dentistry
Medical Innovations
The Apothecary
The Industrial Revolution
Athletics with Safety
American Dentistry
Health and Education

The series of talks was sponsored by the Eastman Kodak Company. No advertising, however, was included in the broadcasts nor any reference to the Eastman Kodak Company except mention of the name of the sponsor.

SITUATIONS WANTED

WANTED—Salaried appointments for Class A Physicians in all branches of the Medical Profession. Let us put you in touch with the best man for your opening. Our nation-wide connections enable us to give superior service. Aznoes National Physicians' Exchange, 30 North Michigan, Chicago. Established 1896. Member The Chicago Association of Commerce.

WANTED—Locum Tenens work; or Arizona affiliation by M.D. qualified to do medicine, surgery, and Physical Therapy. Address E. C. Wills, M.D., Wickenburg, Arizona.

TRAINED NURSE-STENOGRAPHER desires position in physicians office, clinic or hospital historian.
Box B, Care of Southwestern Medicine.

SUPPRESS PUTREFACTION
WITH A FOOD
LACTO - DEXTRIN
(Lactose 75% — Dextrine 25%)
Cooperates with Nature—provides the right soil for the growth of a normal intestinal flora.
Samples and literature on request
The Battle Creek Food Co.
Battle Creek, Michigan

Mercurochrome
220 Soluble
in
OBSTETRICS

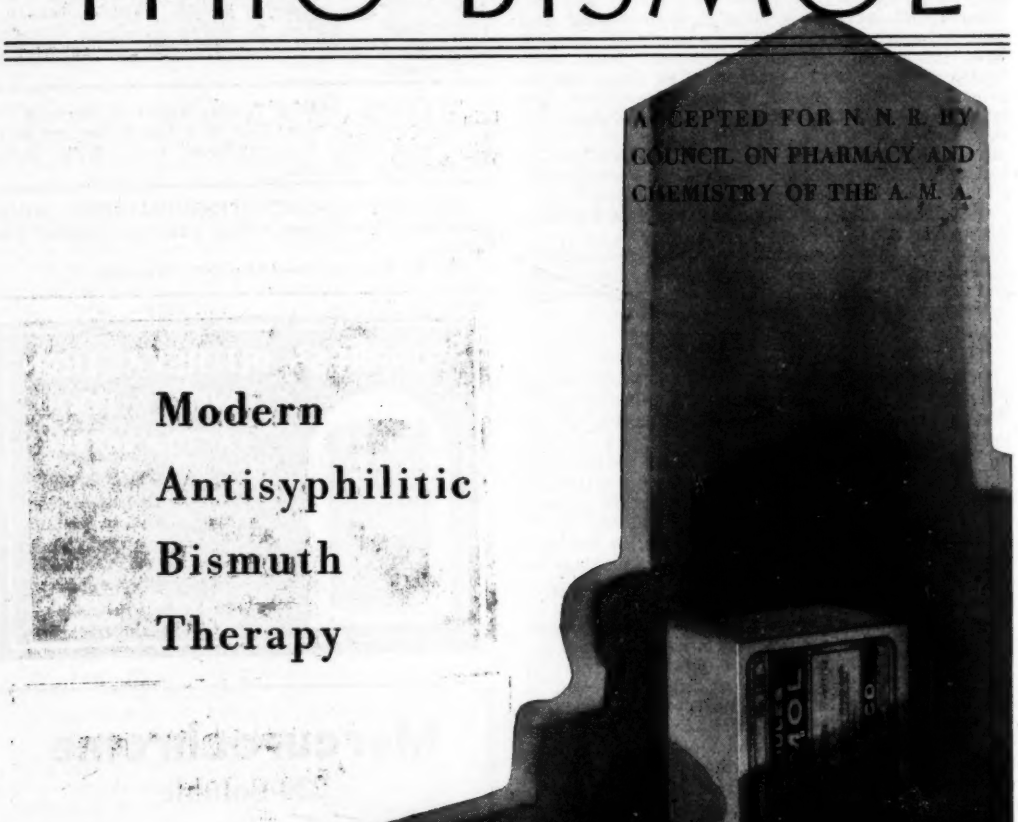
A statistical study of a series of over 9000 cases showed a morbidity reduction of over 50% when Mercurochrome was used for routine preparation.

Write for Information
HYNSON, WESTCOTT & DUNNING, Inc.
Baltimore, Maryland

THIO-BISMOL

ACCEPTED FOR N. N. R. BY COUNCIL ON PHARMACY AND CHEMISTRY OF THE A. M. A.

Modern
Antisyphilitic
Bismuth
Therapy



PARKE, DAVIS & COMPANY

The World's Largest Makers of Pharmaceutical and Biological Products

DETROIT — NEW YORK
CHICAGO — KANSAS CITY
ST. LOUIS — BALTIMORE
NEW ORLEANS — MINNEAPOLIS
SEATTLE

In Canada:
WALKERVILLE — MONTREAL
WINNIPEG